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STREPTOCOCCUS MENINGITIS OF OTITIC ORIGIN; OPERATION, WITH RECOVERY.*

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Otitic meningitis still remains the opprobrium of otology. This is made even more conspicuous by reason of the fact that all the other intracranial complications of otitic origin have slowly but surely yielded themselves more or less to the control of the otologist. In the case of lateral sinus thrombophlebitis the mortality rate has been considerably lowered. Otitic brain abscess is no longer an invariably fatal disease. Osteitis of the petrous pyramid is now a clinical entity in which recovery takes place in the majority of instances. In the main, a more or less universally accepted surgical technique has been evolved and adopted in the treatment of each of these complications. It is not so with otitic meningitis. Our inability to cope satisfactorily with this exigency is at once a striking example of the limitation of surgical practices in this field. Due to a wider knowledge of the many phases of the disease and a better understanding of the pathological processes involved, with special reference to the varied pathways through which infection gains access to the subarachnoid spaces, the many mysteries enveloping otitic meningitis have to a great extent been dispelled. Nevertheless, the many

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years consumed in painstaking research and study of this most important problem, by investigators the world over, have not been rewarded with the evolution of any definite surgical procedure which can assure us of its efficacy even in the smallest proportion of cases. In an effort to cope with this exigency, no less than 10 procedures have been advocated in the treatment of meningitis complicating otitic disease.[1, 2, 3, 4, 5, 6, 7, 8, 9, 10] It is not my intention to enumerate them or to evaluate their individual merits. I can only comment to the extent that in each instance where a particular procedure was recommended, the author substantiated his recommendation by actual recoveries; however, with all these various methods offered for the treatment of otitic meningitis, recovery, when organisms are actually demonstrated in the cerebrospinal fluid, is exceedingly rare. From this fact alone one is justified in questioning the actual specificity or rather the efficacy of any of the methods advocated — to be offered for universal adoption.

It is my belief that recovery has taken place where it has because of some factor which is common to all these cases, rather than to the treatment instituted. In regards to this so-called factor, I have in mind a special strain of organism, perhaps, or a similar pathological state, *e.g.*, where the process is well localized and walled off; or the presence of some substances in the spinal fluid of these patients which is particularly suited to digest and destroy the invading organism.

Whatever plan is outlined in the therapy of a given case of otitic meningitis, one should bear in mind those fundamental surgical principles which govern the treatment of suppurative processes in other localities. This resolves itself, therefore, into a thorough and complete eradication of the original focus and an attempt to promote the elimination of the suppurative elements beyond the original focus by frequent lumbar drainage.

The case herein reported had streptococci in the cerebrospinal fluid. This was demonstrated repeatedly. Although I did a wide subtemporal decompression as soon as I found the increased cell count, I cannot feel that this measure alone was entirely responsible for the child's recovery. Lumbar taps were performed often, and large quantities of fluid were removed. No other medications were employed and the child recovered.

Case Report: L. C., male, age 7 years, was admitted to the Beth Moses Hospital on Nov. 21, 1933, with a diagnosis of an acute surgical mastoid (left).

Previous Personal History: Irrelevant.

History of Present Illness: Sixteen days prior to admission the patient developed an acute suppurative otitis media, complicating a general grippe infection. Forty-eight hours later the drum membrane was incised by an otologist. Relief was obtained immediately after myringotomy. The ear discharged profusely for the next 10 days. During this period there was little pain experienced and the temperature gradually reached normal. On the fourteenth day of the otitis the child began to complain of headache and spent two uncomfortable nights. The temperature rose to 103° in the evenings and the discharge increased in amount. Examination in my office on Nov. 21 disclosed a profusely discharging middle ear, some narrowing of the canal, mastoid tenderness and a temperature of 104°. The child complained of pain above the right eye. This seemed to be the site of his headache for the past 48 hours. X-ray of the mastoids showed the following: Right: large, extensively pneumatized, clear and well outlined. Left: similar in structure but cloudy throughout with evidence of disintegration at the tip and cells about the antrum. Anterior sinus limit 1 in. posterior to canal wall.

Mastoid Operation: Nov. 21. The mastoid was completely broken down. The cells extended in all directions. There was a far forward group in the zygoma, cells up in the squama, unusually large tip cells, a pronounced group of retrofacial cells and a wide, deep Citelli angle. Both sinus and dural plates were visualized and appeared normal and unbroken. Pus taken from the mastoids at operation showed on smear, very many pus cells and Gram positive cocci in chains; on culture, streptococcus hemolyticus.

The postoperative course for the first five days was uneventful. The temperature reached normal on the third day. The wound was clean, sutures were removed and primary union was in evidence.

On the evening of the fourth day, I was notified that the patient had a sudden rise in temperature to 105°. He was

apparently comfortable, complained of no pain, had not vomited and presented no neurological signs. In a room close by, also under my care, I had a child with a postoperative erysipelas and, for the moment, I was fairly convinced that this patient had also contracted the disease. His wound was clean and the skin adjacent was slightly reddened, but owing to the fact that he was an unusually dark complexioned child, I felt that possibly the demarcation would be better demonstrated the next day. A blood culture was ordered and taken. Blood count showed: Hemoglobin, 68 per cent; R.B.C., 3,100,000; W.B.C., 12,000; polys., 72 per cent. Urine was negative.

Nov. 26, 1933 (five days after operation): The patient's temperature dropped in the morning and did not go above 102° for the 24 hours. He had no chills, complained of no headache and seemed apparently in fine spirits. The mastoid wound was clean, with no evidence of any erysipelas. The blood culture of Nov. 25 was reported negative after 24 hours.

Nov. 27, 1933: The condition of the patient seemed unchanged. His temperature reached 102.2° at 4 P.M., and 105° at midnight. He complained of chilly sensations during the afternoon and severe headache over the left frontal region. Vomiting occurred twice during the day. A spinal tap disclosed clear fluid under slightly increased pressure. Cell count was five and six cells per field. Albumin was 1+, globulin negative and sugar was not determined. Both smear and subsequent culture failed to demonstrate the presence of organisms. A blood culture was again taken.

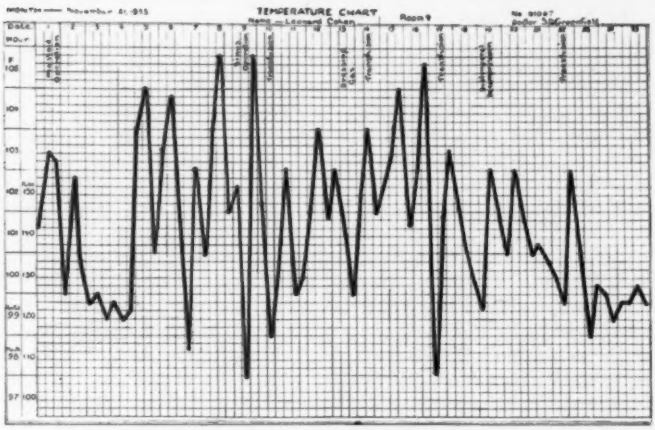
Nov. 28, 1933: Early this A.M., the temperature dropped suddenly from 105° to 97°, and again rose at 4 P.M. to 103.6°. The general condition of the patient seemed changed. He was restless and lapsed off into periods of drowsiness. The blood culture taken on Nov. 25 was reported negative for 72 hours and the one of Nov. 27 was negative for 24 hours. Another culture was taken. Blood count showed little change except for a drop of the hemoglobin to 55 per cent. There was no vomiting and there was absence of all neurological signs.

Nov. 29, 1933: The temperature reached 106° at 4 P.M. He complained of pain in both knees and left forearm. There was no evidence of knee joint invasion.

Ophthalmological Examination by Dr. Bailey: Revealed the absence of ocular palsies and nystagmus. Fundus examination showed both discs hyperemic, retinal veins moderately distended, no hemorrhages, no exudates.

Conclusion: No ocular evidence of any intracranial complications.

Pediatric Consultation by Dr. Gingold was as follows: General condition of patient critical. The throat showed congestion and some postnasal discharge. Heart disclosed a systolic at apex, transmitted posteriorly with fair muscular quality.



Lungs were negative. Abdomen was distended. The liver was one finger below costal margin and the spleen was unpalpable. Extremities showed tenderness upon deep pressure generalized. (No bony or joint involvement.) No petechiae seen.

Impression: Sinus Thrombosis. It seemed at this stage we were dealing with a blood stream infection. With a temperature fluctuating so widely and no other tangible cause to explain the pyrexia, one was justified in venturing such an opinion; however, in the absence of other confirmatory signs, such as a demonstrable bacteremia, absence of definite fundus changes and no splenic enlargement, I felt there was no ur-

gency in exploring the lateral sinus that day and decided to wait another 24 hours.

The hemoglobin on Nov. 29 was 62 per cent. W.B.C. was 18,400, with 82 per cent polys.

Nov. 30, 1933: The temperature rose to 107° at 4 P.M., and the child appeared critically ill. The blood culture of Nov. 27 was negative after 72 hours. However, in view of this wide fluctuation in temperature, despite the negative blood culture, I deemed it advisable to explore the lateral sinus.

Operation (Nov. 30, 1933): Upon removal of the sinus plate, which appeared intact and apparently normal, the sinus wall itself showed congestion. The latter was normal in color and there was absence of thickening. Some fine granulations seemed visible on that portion of the exposed sinus in the direction of the bulb. These became more pronounced when the sinus wall was depressed away from its bony covering; therefore, I felt that there might be something in the bulb and I proceeded to close off the sinus at both ends and incised its wall. This was followed by free bleeding from both the torcular and bulbar ends. The sinus was then packed off and the wound closed. Owing to the fact that I found so little pathology in the sinus I did not ligate the jugular vein.

Dec. 1, 1933: There was little general reaction following exploration of the sinus. The temperature dropped in the morning to 99.6° and rose to 102° in the evening. The patient received a transfusion of 300 cc. by direct method. The blood culture of Nov. 28 was reported negative after 72 hours and another one was taken.

Dec. 2-8, 1933: During this period the temperature fluctuated widely. Some days it ranged between 100° and 103°, and on others it reached 105°. Patient complained occasionally of headache, moderately severe in character and located over the frontal region. At no time did he present any neurological signs. He did not vomit and took nourishment well. Repeated fundus examinations were negative. Wound was dressed at regular intervals and nothing unusual noted. On Dec. 5, six days after exploration of the sinus, the incision in the sinus wall was closed and upon removal of the plugs no bleeding occurred. No effort was made to drain the interior

of the sinus. The patient was again transfused on Dec. 4 and Dec. 7. Blood cultures taken on Dec. 4 and Dec. 8 were subsequently reported negative.

Dec. 8, 1933: The proceeding night was marked by a distinct change in the patient's condition. He complained bitterly of headache, was unusually restless and vomited several times. His temperature reached 104° at midnight. As I approached his room I was struck by his cries, which seemed to be typically meningeal. Examination revealed some slight neck rigidity and a left Kernig. A lumbar tap disclosed ground glass fluid under pressure. Cell count was 1,400 W.B.C., with a differential of 84 per cent polys. and 16 per cent lymphocytes. Albumin was 3+, globulin 2+, and sugar, 55. Smear showed many pus cells but no organisms. Culture of this fluid was negative after 72 hours.

Dec. 9, 1933: The patient's condition seemed somewhat improved. His headache was relieved after the tap and he appeared much brighter. He was seen in consultation with Dr. Friesner and Dr. Moorhead. Examination at this time disclosed a questionable neck rigidity and the Kernig of the day before was not present at this time. Eye grounds were negative. After deliberation no operative procedure was advised, but in the absence of any neurological signs and the apparent good condition of the patient, one could not reconcile these clinical findings with those of the report of the spinal fluid of the day before; therefore, it was decided to follow the expectant plan of treatment.

Dec. 10, 1933: On this day the patient again began to complain of headache. His temperature reached 103° in the morning. He was restless and irritable. He vomited several times and refused to take food. Neck rigidity was definite and a bilateral Kernig was present. Lumbar tap released 25 cc. of cloudy fluid under increased pressure. Cell count showed 2880 W.B.C., with 85 per cent polys. and 15 per cent lymphocytes. Albumin 4+, globulin 3+, and sugar not determined. No organisms were found on smear. Culture, however, showed the streptococcus hemolyticus after 48 hours. With these findings I decided to do a subtemporal decompression with the hope of finding some epidural collection of pus.

Operation (Dec. 11, 1933): The old mastoid wound, which had practically healed, was reopened. The dural plate was

removed from the floor of the middle fossa forward into the zygoma, over the squama and the angle between sinus and dura. I did not drain the basal cisterna. No epidural collection was found. The dura seemed fairly normal throughout. I elevated the latter off the superior and posterior surfaces of the petrous pyramid, but did not disturb the petrosa itself. A lumbar tap was again performed immediately after the operation and all the fluid that would drain off was removed.

Dec. 12, 1933: The patient's condition seemed to be far better than was expected. His temperature dropped to 101° and he appeared fairly comfortable. Headache had entirely disappeared and vomiting ceased. A lumbar tap was performed and 25 cc. of cloudy fluid was removed.

Dec. 13-16, 1933: During this period the patient received no medication. He complained of occasional headache. His neck rigidity, which was never very marked, was barely noticeable. All other neurological signs were absent. Lumbar taps were performed daily and large amounts of fluid were withdrawn.

Dec. 17, 1933: The temperature reached 105° this day, but the patient had little headache. There was no vomiting. The neck rigidity was definite, but all other neurological signs were absent. He was seen in consultation with Dr. Josephine B. Niel. Her impression was that we were dealing with a localized meningitis and she ventured a favorable prognosis. Lumbar tap showed cloudy fluid under slight pressure. 14 cc. were removed. W.B.C., 4000; polys., 82 per cent; lymphocytes, 18 per cent; albumin, 4+; globulin, 3+, and sugar, 40. Smear, many pus cells and gram positive diplococci. Culture, streptococcus hemolyticus.

Dec. 18-22, 1933: During this period and thereafter the temperature did not rise above 100°. The patient complained of occasional headache. He ate well, gained weight and seemed comfortable. The taps performed on these four days were as follows:

Dec. 18, 1933: W.B.C., 1400; polys., 78 per cent; lymphocytes, 22 per cent; albumin, 2+; globulin, 1+; sugar, 45; smear, no bacteria; culture, streptococcus hemolyticus.

Dec. 19, 1933: W.B.C., 900; polys., 74 per cent; lymphocytes, 22 per cent; albumin, 3+; globulin, 2+; sugar, 40; smear, no bacteria; culture, streptococcus hemolyticus.

Dec. 20, 1933: W.B.C., 450; polys., 74 per cent; lymphocytes, 20 per cent; albumin, 2+; globulin, negative; sugar, 45; smear, gram positive cocci; culture, negative after 72 hours.

Dec. 22, 1933: W.B.C., 160; polys., 62 per cent; lymphocytes, 38 per cent; albumin, 2+; globulin, negative; sugar, 55; smear, gram positive cocci; culture, negative after 72 hours.

Dec. 22, 1933-Jan. 5, 1934: During this period the patient's condition rapidly improved. He ate well, slept well and gained weight. His posterior wound granulated rapidly and his middle ear was dry. A tap performed on Dec. 26 showed: W.B.C., 170; polys., 20 per cent; lymphocytes, 80 per cent; albumin, 1+; globulin, negative; sugar, 60; smear, no bacteria; culture, negative after 72 hours.

Tap on Jan. 3, 1934: W.B.C., 60; all lymphocytes; smear, no bacteria; culture, negative after 72 hours.

Patient left the hospital Jan. 5, 1934. He continued dressings at my office and was discharged as cured on March 22, 1934.

CONCLUSIONS.

1. The case herein reported is unquestionably one of streptococcus meningitis. The organism was found both on smear and culture in the same specimen of spinal fluid on two occasions and in either smear or culture in six other specimens; running parallel with the disappearance of the organism, the cellular elements diminished and the sugar content increased.

2. It is a most difficult task to define with any degree of certainty what actual pathological processes were involved in this case. It is reasonable to assume, however, that in view of the fact that the patient recovered, the meningeal involvement was most assuredly not a diffuse one; furthermore, in the absence of more pronounced neurological signs, particularly the muscle rigidity, the process must have been well localized and limited essentially to the middle fossa. Why the

infection became localized and remained so is left for one to conjecture.

3. To trace the avenue of invasion from the seat of the original infection in the mastoid to the meninges must be done purely upon a speculative basis. Bony extension by way of the pyramid does not appear probable. This patient presented none of the signs and symptoms of petrositis. There was no post-operative period of quiescence to speak of, followed by one of low grade sepsis. He never complained of retrobulbar or supraorbital pain. His middle ear was dry immediately after the mastoid operation and remained so throughout his illness. It is perhaps more logical to look to one of the venous channels as the avenue along which the infection was propagated to the meninges. The temperature on the fourth postoperative day began to assume the typical septic character. There was an increased leukocytosis (22,000) and a low hemoglobin (55 per cent). The patient complained of chilly sensations on several occasions and at one time had generalized joint pains. All these are typical of blood stream infection despite the fact that a bacteremia could not be demonstrated. The failure to obtain the latter substantiates even further the local character of the entire process. Which venous channel was responsible for carrying the infection is again left to speculation.

4. A very interesting bacteriological feature was the actual demonstration under the microscope of the degeneration of the organisms. The first positive specimen of the cerebrospinal fluid showed the streptococcus occurring in long chains, rounded and well outlined, taking the stain very intensely. In the later specimens the chains were shorter, not as abundant and the organisms appeared paler. In the last positive fluid the chains were all gone, the organisms were few; were found isolated, enlarged and swollen, taking the stain very poorly. There was evidently some substance in the cerebrospinal fluid that was peculiarly potent to cause the destruction of this particular strain of streptococcus.

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EMPYEMA OF LATERAL SINUS.*†‡

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Empyema of the lateral sinus is a condition brought about by the liquefaction necrosis of an infected thrombus. This type of lateral sinus disease is of special interest because it demonstrates the role which nature plays in inflammatory diseases of the lateral sinus, the correct interpretation of which makes for more accurate diagnosis and more effectual therapy. A resumé of the etiology and pathology of lateral sinus phlebitis and thrombosis might be of value before proceeding with a report of a typical case.

Braun[1] enumerates the following causes of sinus phlebitis:

1. Extension from the longitudinal sinus, due to scalp infection.

2. By contiguity from a cervical adenitis.

3. From middle ear disease, as: *a.* an extension of thrombophlebitis of the tympanic veins; *b.* coalescence of the hypotympanic and peribulbar cells.

4. From mastoid disease, as follows: *a.* a diseased inner table, producing a perisinus abscess, then a periphlebitis, the result of which may lead to a thrombosis; *b.* inner table disease may produce an intravenous thrombosis without objective periphlebitis or the formation of a perisinus abscess; *c.* extension of thrombi or phlebitis from the mastoid veins which empty into the sinus at the knee; *d.* from a petrositis—by extension of either or both of the petrosal sinuses.

The case cited in this discussion is an example of Class 3, in which the patient's symptoms were out of proportion to the objective ear findings, and were due to an extension of thrombophlebitis of the tympanic veins to the bulb. This was further substantiated by the absence of pus and mastoid de-

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†From the Otolaryngological Department of Cumberland Hospital.

‡Preoperative diagnosis can be made, based on signs suggestive of both sinus thrombosis and brain abscess.

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struction at the time of the simple mastoidectomy, and also by the appearance of good sinus plate and objectively good sinus vein wall, when the lateral sinus was incised at the second operation.

The possible courses a lateral sinus phlebitis may take can be enumerated as follows:

1. The phlebitic area may be walled off by a mural thrombus and recovery take place.

2. The phlebitis may spread, with little or no attempt at localization or thrombosis. Death usually results from general sepsis. This may occur even if an obliterating thrombus does form.

3. The phlebitic area may be walled off by a thrombus which later undergoes liquefaction necrosis. This may cause a leak through the visceral layer, forming a cerebellar abscess; or it may break through the outer layer and drain, or communicate with a perisinus abscess or mastoid. If no surgical tract for drainage is established, the pus may drain along the mastoid emissary and form a fluctuating mass under the scalp. Drainage through the posterior condyloid foramen is also possible.

4. The thrombus may become organized, with the resultant obliteration of the sinus; or there may be absorption of part or all of the thrombus, with recanalization of the sinus.

This case took the course noted in Class 3; however, drainage was instituted before leakage occurred, through the walls of the lateral sinus. Harry Friedenwald[2] reports two cases of empyema of the lateral sinus which were improving clinically at the time of operation, at which time he found the outer wall of the lateral sinus eroded, thus demonstrating nature's efforts to cure septic thrombosis of the lateral sinus. L. Baer[3] reports a case of lateral sinus thrombosis, in which a fluctuant mass developed above an almost healed mastoid scar, due to an empyema of the lateral sinus, causing an erosion of the outer wall. H. H. Martin and S. J. Crowe[4] report a case of lateral sinus thrombosis, in which an empyema was discovered, causing an erosion of the inner wall, with the formation of a cerebellar abscess. E. M. Seydell[5] reports a case of lateral sinus thrombosis, with erosion

through the outer cortex, the sinus having been opened and drained through a skin incision, which later required more complete operation, with recovery.

The clinical picture of lateral sinus disease does not follow a definite course. G. L. Tobey, Jr.[6] divides his cases clinically into three groups:

1. The septic, explosive type, with acute onset and a picture of sepsis, with sudden remissions of temperature.

2. The typhoidal type. Here the temperature shows persistent irregularity of from one to two degrees, with slight remissions.

3. The latent type, with slight or no rise in temperature.

Dr. Tobey based his diagnosis on evidence of sepsis and signs and symptoms of meningeal irritation. Later he and J. B. Ayer[7] brought out the value of the spinal fluid pressure variation when the internal jugular veins were compressed. The presence of an obliterating thrombus causes no variation in the pressure, while the patency of the vein causes a rise.

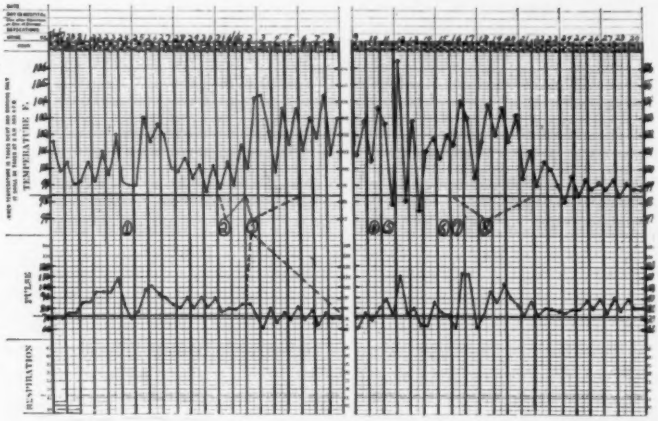
Braun states that a sinus phlebitis or thrombosis may cause alterations in brain tissue or the meninges by direct extension, or by involving the pial and cerebral veins, with formation of spots or hemorrhagic softening in the brain. Aphasia and agraphia may occur, due to lesions in the medulla or due to thrombi in the larger pial vessels surrounding the temperosphenoidal lobe, thus producing an edema. If this edema includes the meninges, the cranial nerves may be directly involved, or secondarily involved as a result of arachnoid effusion.

While the literature contains many reports of cases in which sinus phlebitis and thrombosis were accurately diagnosed and successfully treated, there is no report, to my knowledge, in which a preoperative diagnosis of empyema of the sinus was made. The clinical picture and laboratory data in the case herein cited were suggestive of a sinus thrombosis and of a cortical brain abscess. The therapy was based on the understanding that nature tries to wall off infection.

A report of the case follows:

T. E., male, age 39 years, was admitted to the hospital on May 19, 1932, complaining of severe pain in the left ear, pain

over left mastoid region and severe vertex headache. The duration of his illness before admission was five days. Temperature on admission was 101.4° F. Examination showed a dull gray drum slightly thickened, with the landmarks still visible. There was moderate canal wall sag, mastoid blurring and tenderness. A myringotomy liberated a moderate amount of seropus. Because of persistence of temperature and the vertex headache becoming more severe, a simple mastoidec-



(1) Simple mastoidectomy. (2) Slight rise in temperature; first indication of possible complication. (3) Sharp rise in temperature, with all meningeal symptoms and drop in pulse rate; however, the meningeal symptoms became less marked in the following three days. (4) Patient became drowsy, indicating a breaking down of clot with adjacent brain or arrachnoid edema. (5) Internal jugular ligation and lateral sinus drainage with postoperative temperature excursion, indicating embolic phenomenon. (6) Return of drowsiness, indicating pus in torcular, (7) which was opened and drained. (8) Temperature due to previous embolic phenomenon, mediastinal metastasis, and drainage into bronchus.

tomy was performed on May 24, 1932, the tenth day of the illness. There was no frank pus in the mastoid, although all the cells showed marked congestion of the mucosa. The mastoid was well exenterated down to a good table over both tegmen and sinus. No exposures of sinus or dura were made. There was the usual postoperative reaction, with a return of the temperature to normal on the sixth day. On June 2, or the ninth postoperative day, the temperature rose to 104.2° F., associated with chilliness but no actual chill. With the

rise in temperature, the pulse dropped to 72. The following two days the patient became worse, his temperature rising to 104.4° and the pulse dropping to 60. The patient complained of severe vertex headache, photophobia and scalp neuralgia. Nuchal rigidity, positive Kernig and right Babinsky were present.

At this critical stage the sepsis and meningeal sinus suggested sinus phlebitis. Brain abscess (secondary) or meningitis were also considered. Because the original mastoid focus had been so thoroughly removed, it was judged that little could be gained by revision. More time for nature to localize whatever was taking place was thought advisable, before any intervention should be considered. A blood culture was taken. No spinal tap was done, as we are generally reluctant to tap the spine when there seems to be a leak into the circulation, and the possibility of a brain abscess was also considered in this instance. The patient was typed and donors prepared, as frequent small transfusions have proven the greatest help in fighting phlebitis.

The following three days brought a marked improvement. The patient's temperature came down to 101° F., and the meningeal signs began to subside, so that flexion of the neck was possible. The laboratory reported the first blood culture positive for streptococcus hemolyticus. The subsequent cultures were sterile. With the marked improvement and lack of evidence that there was any more leaking into the blood stream, further observation was deemed advisable. General support with regard to fluid intake, transfusions, alkalinization, etc., were carefuly carried out. With the pulse still slow, brain abscess, in addition to the sinus disease, had to be considered, and a spinal tap was not done for fear of disturbing things intracranially. On June 10th, eight days after the sudden rise in temperature, the patient became very drowsy. There were no localizing signs, and the earlier signs of meningeal irritation were rapidly subsiding. However, an indefinite right Babinsky and a poorly sustained ankle clonus were still present. The eye grounds were normal. A spinal tap was done at this time. Great care was exercised to prevent the fluid from gushing out, and very little was removed. The cell count was 60. Compression of the left internal jugular caused no rise in pressure. Compression of the right internal jugular caused a rise of 12 mm. Hg.

The positive blood culture, and the Tobey-Ayer test, with evidence of no blood in the left internal jugular, pointed to a thrombosis of the left lateral sinus. The slow pulse, cortical headache, drowsiness and increased cell count in the spinal fluid were suggestive of a brain abscess. It was judged that if the thrombus broke down and formed an empyema in the sinus, the findings would be those of a thrombosed sinus and a cortical abscess (for an empyema of the sinus can be considered an intracranial abscess). Of course, the possibility of a metastatic abscess was also considered, as well as the possibility that the slow pulse might be due to stimulation of the vagus and trigeminal, as occurred in a case of lateral sinus thrombosis reported by C. B. Holmes and H. M. Goodyear.[8]

Operative procedure, performed by the writer on June 11, nine days after onset of sepsis, was as follows: The internal jugular vein was ligated under local anesthesia. Under gas-oxygen the plate over the sinus was removed. The sinus wall was slightly off-color, with no granulations on it. Incision into the sinus produced a flow of pus and broken down granulations. The wall was resected from the bulb to within a half inch of the Torcular Herophili. No blood flow was obtained.

The following day there was a rise in temperature to 106.4° F., due to metastasis to a mediastinal gland, which later ruptured into a bronchus. The evidence of this was in the X-ray findings and also was indicated by the fact that the patient coughed up blood and some pus, and complained of pain over the cardiac region.

Five days after the operation the patient again began to be drowsy. The writer again operated upon the patient, this time opening the torcular. A very small amount of pus was found in it, with no return flow of blood.

Following this operation, the patient made an uneventful recovery. No further operative procedure was necessary other than suturing the gaping wound. The patient received an average of two transfusions a week during the critical period.

SUMMARY AND CONCLUSIONS.

A review of the literature of causes, pathology and clinical courses of lateral sinus disease.

A typical case of empyema of the lateral sinus, in which a preoperative diagnosis was made, based on clinical and laboratory findings, pointing to occlusion of the lateral sinus, sepsis and signs of cortical brain abscess.

Attention is called to the role nature plays in localizing infection, and to the wisdom of intervening only after nature has been given a chance.

The writer wishes to express his thanks to Dr. Frank H. Lasher for the privilege of reviewing and reporting this case, which occurred on his service.

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ELECTRO-ACUSTO-TESTOMETER FOR ACCURATE TIMING AND RECORDING OF BONE CONDUCTION TEST.

DR. M. MARTYN KAFKA, Brooklyn.

Sound is the physiological phenomenon of sensation induced in the mechanism of hearing by the vibratory motion of matter. Sound vibration intensity varies. The disturbance of bodies by any form of impact or friction produces vibration in the molecules of such bodies; this results in sound. It has been observed that the velocity of sound is dependent on three factors: elasticity, density and the molecular structure through which the medium is transmitted. Variations in the quality or pitch of sound depend upon the regularity and the rapidity of the vibrations.

We are aware that any one part or even all parts of the conducting apparatus of hearing may become pathologic and it is the purpose of hearing tests to differentiate, diagnose and localize the nature and the extent of the lesion. The efficient and normal interpretation of sound is dependent upon the perfect physiological hearing.

The tuning fork test possesses a special diagnostic value and by comparison of the conducting power of the cranial bones with that through air, the examiner may differentiate labyrinthine affections. In order to accurately determine the condition of the auditory function, repeated and exact tests must be made, uniform results being necessary for definite and reliable, scientific conclusions.

I have found in my experience that the diagnostic procedures as related to hearing tests have not been very accurate. It has been my aim to present to the profession an instrument which will accurately measure and time the most commonly used bone conduction diagnostic tests as related to hearing.

This instrument is, essentially, based upon the Schwabach bone conduction test and related tests of Rinné and Weber.

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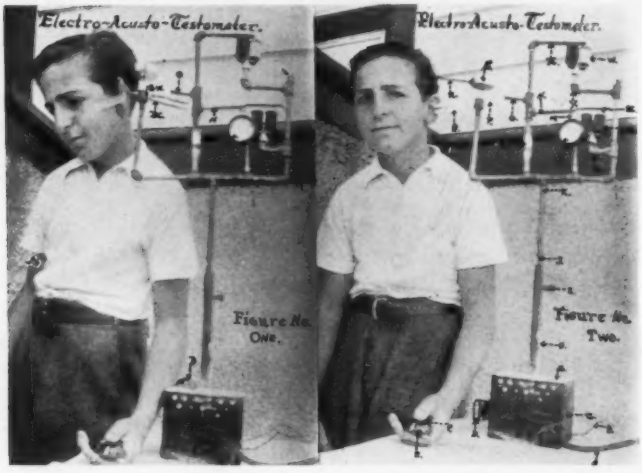

Fig. 1. (D) Push-button used by the operator for starting the vibration of the tuning fork (B.C.) and simultaneously pushing the stem of the stop-watch down (M, Fig. 2). (L) Stop-watch, enclosed in a metal case with face of watch facing operator. (N) Electrically controlled lever for pushing down stem of stop-watch. (O) Electrically controlled lever for striking tuning fork, which is held in position for recording the bone conduction. (B.C.) Tuning fork as it appears for bone conduction test. (S.W.) Insulated joint swivel for holding tuning fork (256, D.V.) in position.

Fig. 2. (A) Thick coil (electrical wire) for current from wall. (B) Electrical wire united to (D) push-button for operator's use. (C) Heavy base stand of the electro-acusto-testometer. Within the base is placed the transformer which changes the voltage from 110 to 5 volts. (D) Push-button for operator's use in controlling electrically the levers (O) and (N). (E) Push-button whcih the patient can operate to stop electrically the stop-watch (L) without interfering with the vibrations of the tuning fork (Q). (F) Thumb of patient pressing the push-button down. (G) Upper surface of the base of the instrument. (H) Upright bar through which wires are passed to the electro-magnets (V) and (W). (I) Screw for adjustment of the instrument height (according to height of patient). (J) Bar which can swing the instrument in any direction for the convenience of the patient or the operator. (K) Junction where the wires running to electro-magnets pass in the direction of (V) and (W). (L) Stop-watch. (M) Stem of stop-watch. (N) Electrically controlled lever to push stem of stop-watch down. (O) Electrically controlled lever to strike tuning fork only when tuning fork (Q) is in position for bone conduction test, otherwise as it is in this position for the Rinné Test the tuning fork is, with the aid of the part (S.W., Fig. 1) swivel—rubber insulated—changed from the bone conduction position to air-conduction position. (P) Hilt of tuning fork as it projects from the rubber insulated swivel. (Q) Tuning fork held in a position for testing the air conduction. (R) Steel ruler (roll) for measuring accurately the distance the tuning fork is held from the auricle. (S) Steel ruler roll in position. (T) Spring for swinging the armature electrically by means of the electro-magnet's power (W). (U) Joint through which electrical wires are communicated to electro-magnet (V). (V) Electro-magnet which operates independently the lever (Z), which can press the stem of stop-watch down without affecting the fine vibrations of the tuning fork. (W) Electro-magnet which controls the armature to which is attached the two levers (N) and (O). (X) Armature which operates when push-button (D, Fig. 1) is operated. (Y) Joint through which electrical wires are united to electro-magnet from transformer. (Z) Armature which controls the lever for pressing down the stop-watch stem (M) and which operates when the patient pushes the push-button (E).

At present, the diagnostic tests of hearing are performed "by hand." The variations that are recorded in this manner are not precise and uniform, since the blow struck the tuning fork varies with each performance. Also, the tuning fork is not held at the same and exact distance from the ear each time the Rinné test is performed. The designation of the terms "positive" or "negative" Schwabach or Rinné are at best approximate, since no precise time as to the number of seconds "negative" or "positive" are given. It also may be that the examiner, himself, may have defective bone conduction when he compares his bone conduction with that of the patient. Then again, the examiner's hand absorbs some of the vibrations, especially if the fingers are placed near the nodal point of the fork. Another disadvantage is that the patient, during examination of his hearing, usually signals the examiner by raising or lowering his hand, thus some perceptible time is lost. Due to these variations, reports submitted by different otologists and institutions vary.

With the aid of this instrument the results obtained will always be constant. The blow struck the tuning fork will be with the same force as it is controlled by an electro-magnet which is operated by the patient himself by means of a push-button which is attached to electrical wires. Not only does the stop-watch record simultaneously the actual time the sound is heard through bone conduction, but the patient himself can, by means of another push-button, stop the watch when he no longer hears the vibrations, either through bone conduction or by means of air conduction when the Rinné test is performed. The tuning fork does not have to be compared to the examiner's ear, as the mean average of the number of vibrations is already recorded for normal.

The fork is suspended in a rubber insulated clamp and is stationary. The point where the tuning fork is held is fixed and does not interfere with the normal cross vibrations at the nodal point. The distance the tuning fork is held from the ear in the Rinné test can be precisely measured by means of a steel roll ruler which is attached to the instrument and can be swung in different directions. The results can be permanently recorded as to the number of seconds "positive" or "negative" for Schwabach or Rinné. All otologists and various institutions can have uniform results for bone conduction tests.

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EXTERNAL OTITIS DUE TO DIPHTHERIA BACILLUS.

DR. E. M. ARNOLD, Houston.

This form of otitis is rather rare, especially when the primary lesion is in the canal. This case occurred in a man, age 30 years and weighing about 170 pounds. As is usual in such cases of primary infections there were very little constitutional symptoms. Patient was not very sick and it was with difficulty that he was kept in the house, his only complaint being some discomfort in the canal and slight deafness.

No history of exposure to diphtheria was obtained, neither was there any sore throat nor nasal discharge.

Examination disclosed an ear canal which had the appearance of fungus infection (aspergillus) and was so diagnosed. On second day, however, when the ear canal was being cleaned it was noticed that the raw surface was hemorrhagic and it was then that the diagnosis of diphtheria was suspected, and the diagnosis was confirmed by a report from the pathological laboratory of Dr. Martha Wood.

The case was reported to the health department and diphtheria antitoxin ordered and 20,000 units were given very reluctantly by the department. The physician stated that evidently there was some mistake as he had never seen a case of diphtheria of the ear.

Local treatment of 50 per cent alcohol was kept up but no attempt was made to remove the membrane. The drum was not perforated and there was very little discharge. Two days after the antitoxin was given anaphylactic reaction took place, temperature of 102°, headache, urticaria, etc. This lasted about two days. Smears were made from the ear periodically, no more antitoxin was given and it was not until Sept. 5 that the patient was discharged from the quarantine.

Ear is normal at this time and the drum shows no evidence of perforation.

1215 Walker.

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THE CHEMICAL COMPOSITION OF SECRETIONS FROM ALLERGIC RHINITIS AND SINUS EXUDATES.*†

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Chemical analyses of nasal and maxillary sinus secretions and exudates in cases of allergic rhinitis and maxillary sinusitis were made with the idea that the findings might throw some light on the pathological changes in the mucous membrane. The cases of allergy selected had the usual pale, boggy, edematous mucous membrane, profusely discharging a mucinous secretion containing many eosinophilic leukocytes. The cases of suppurative sinusitis had positive Roentgenographic findings, were dark upon transillumination and showed the usual symptoms of sinusitis. Smears of the exudate showed many polymorphonuclear leukocytes, monocytes, lymphocytes and epithelial cells.

The nasal discharge from allergic patients was collected in chemically clean glass-stoppered bottles and brought to the laboratory on the same day. The exudate from suppurative maxillary sinusitis was aspirated into a syringe through a Dean needle as completely as possible, the remaining material was washed out with distilled water and the returned liquid saved for analysis. (Normal saline was always used following the treatment with distilled water to eliminate as far as possible any swelling of the tissue by the hypotonic solution.)

The material secured was transferred to a tared silica crucible and cover, weighed, evaporated to dryness on a water bath and weighed again. The residue was ashed with concentrated sulphuric acid and the final weight taken. The water content, total solid, ash and protein were calculated from these readings. The ash from the secretions, aspirated and collected, were dissolved in a volume of 0.1 N. hydrochloric acid equal

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to, or twice, that of the original weight, whereas, the ash from the washings was dissolved in 10 times the weight of the total solid. Portions of these solutions were used to determine calcium, sodium and potassium. Calcium was determined by the Clark-Collip modification of the Kramer-Tisdall Method,[1] potassium by the Kerr modification of the Kramer-Tisdall Method,[2] and sodium by the Rourke modification of the Kramer-Gittleman Method.[3] Each method was checked and found to be accurate with error of ± 5 per cent in the range of amounts analyzed. Most of the determinations varied with-

TABLE I. THE COMPOSITION OF NASAL DISCHARGE IN ALLERGIC RHINITIS.

Patient	Water %	Solid %	Ash* %	Protein %	Ash in Solid %	Ca. Mg. %	Na. Mg. %	K. Mg. %
H.A.	97.84	2.16	1.13	1.03	52.4	6.2	324	61
L.H.	98.53	1.47	1.26	0.21	85.5	7.6	323	69
L.H.	98.45	1.55	1.27	0.28	82.2	6.1	275	70
L.H.	98.49	1.51	1.28	0.23	84.7	5.9	274	66
C.E.	98.42	1.58	0.88	0.70	55.8	9.6	214	67
C.E.	95.72	4.28	1.12	3.16	26.1	12.0	232	68
L.B.†	95.48	4.52	1.22	3.30	27.0	21.0	329	80
L.B.	98.02	1.98	1.39	0.59	71.6	14.8	336	97
R.S.	96.20	3.80	1.10	2.70	29.0	10.4	295	90
R.S.	94.95	5.05	1.23	3.82	24.4	12.9	292	76
R.W.	97.22	2.78	1.15	1.63	41.4	9.7	.....	....
E.R.	97.64	2.36	1.28	1.08	54.3	.....	.....	....
E.R.	96.58	3.42	1.24	2.18	36.3	12.2	253	77
E.R.	97.36	2.64	1.43	1.21	55.1	11.0	274	75
E.R.	95.08	4.92	1.10	3.82	22.3	12.3	.....	67
E.K.	97.46	2.54	1.24	1.30	47.8	9.3	354	58
E.K.	98.30	1.70	0.97	0.73	57.0	.....	.....	....
E.K.	97.81	2.19	1.17	1.02	53.3	9.2	318	75
E.K.	97.78	2.22	1.18	1.04	53.2	7.9	301	62
E.K.	97.79	2.21	1.17	1.04	52.8	7.5	.....	83
E.K.	96.27	3.73	1.38	2.35	37.1	13.5	543	89
H.M.	96.33	3.67	1.24	2.43	33.8	9.3	300	65
H.M.	94.99	5.01	1.19	3.82	23.6	12.1	301	66
H.M.	95.53	4.47	1.27	3.20	28.5	11.3	234	68
H.M.	96.48	3.52	1.29	2.23	36.5	11.3	180	84
H.M.	96.56	3.44	1.07	2.37	30.9	8.2	254	73
H.M.	97.04	2.96	1.16	1.80	39.0	9.6	322	73
R.D.	97.98	2.02	1.25	0.77	61.4	11.0	324	99
E.V.†	95.79	4.21	1.26	2.95	29.6	13.5	218	92
E.V.	92.16	7.84	1.14	6.70	13.9	15.5	245	152
I.T.	98.00	2.00	1.07	0.93	54.0	12.1	276	116
I.C.	98.14	1.86	1.21	0.65	64.5	37.8	339	95
L.W.	97.64	2.36	1.34	1.02	56.6	6.8	202	66
L.W.	96.57	3.43	1.21	2.22	35.3	13.5	297	74
Average	96.90	3.10	1.20	1.90	47.8	11.3	291	79

†Allergic condition with superimposed infection.

*Ash is determined as the sulphate.

The samples vary in size from 0.2968 gm. to 9.9 gm. Only five were below 1 gm. and 21 weighed more than 2 gm.

in an error of ± 2 per cent. Duplicates were made almost always on potassium and on sodium and calcium when sufficient material was available.

RESULTS.

A comparison of Tables I and II shows the following differences in material analyzed.

1. The water content of the nasal discharge from allergic rhinitis is greater than that of suppurative maxillary sinusitis. The averages are 96.90 per cent and 90.10 per cent respectively.

2. The total solids of sinus exudate is about three times that of the secretion in allergy.

3. The ash content of the allergic individual has a smaller range and a slightly higher average than the sinus exudate, the former 0.88 per cent to 1.43 per cent, average 1.20 per cent as compared to the latter values 0.46 per cent to 1.54 per cent, average 1.12 per cent.

4. The protein in secretions from nasal allergy varies from 0.21 per cent to 3.82 per cent in contrast to 3.45 per cent to 16.23 per cent in sinus exudates. The percentage of protein is obtained by subtracting the per cent of ash from the per cent of total solid. These values include the carbohydrates and fats, but they are present in minute quantities in comparison with the protein, that the error involved is negligible. The values here given correspond to the protein values calculated from the nitrogen content in an earlier paper,[4] and are in agreement with the findings of Mittermaier.[5]

5. The ash in the total solid is much greater in allergic rhinitis than in the exudate from sinusitis. In some instances the value is as high as 85.5 per cent of the total solid. This type of secretion from cases of allergy contains very little mucin and only a very few cells. The goblet cells of the mucous membrane are not functioning, probably due to a lack of stimulation or some insuffiency of the gland.

As stated above the secretions from allergic rhinitis contain more minerals than the exudate from maxillary sinusitis. The data show that the greatest difference exists in the calcium values, averaging 11.3 per cent and 8.73 per cent, respectively.

TABLE II. COMPOSITION OF THE EXUDATE FROM SUPPURATIVE MAXILLARY SINUSITIS.

Patient		Water %	Solid %	Ash† %	Protein %	Ash in Solid %	Ca. Mg. %	Na. Mg. %	K. Mg. %
D.K.		92.96	7.04	1.01	6.03	14.4	9.6	.....	12.0
R.H.		86.70	13.30	1.21	12.90	9.1	8.1	237	117
W.W.		91.92	8.08	0.78	7.30	9.7	12.0	267	15.6
R.F.	L.	90.96	9.04	1.46	7.58	16.17	19.0	387	7.2
R.F.	R.	90.35	9.65	0.85	8.80	8.84	10.6	295	....
R.F.	L.	90.98	9.02	1.26	7.77	13.95	10.9	266	38.5
R.F.	L.	93.48	6.52	1.00	5.52	15.40	9.2	304	73.8
M.S.		88.51	11.49	1.11	10.39	9.7	9.2	229	103
M.S.		90.45	9.55	1.00	8.55	10.5	6.5	228	9.6
H.B.	R.	82.60	17.40	1.17	16.23	6.7	24.9	320	....
H.B.	L.	85.20	14.80	1.17	13.63	7.9	25.4	320	....
E.W.		90.46	9.54	1.07	8.47	11.2	9.6	274	87
J.G.		95.86	4.14	0.97	3.17	23.2	9.8	351	126
E.S.		88.62	11.38	1.17	10.21	10.2	5.0	322	104
E.S.		88.10	11.90	1.15	10.75	9.7	4.9	.....	....
S.M.		90.20	9.80	1.00	8.80	10.2	4.4	323	115
S.M.		89.90	10.10	1.18	8.92	11.68	4.2	325	....
S.M.		91.25	8.75	0.94	7.81	10.73	4.6	301	117
S.M.		88.80	11.20	1.15	10.05	10.2	.....	.....	139
S.M.		90.80	9.19	1.00	8.20	10.8	4.0	310	131
S.M.		89.70	10.30	1.05	9.25	10.2	4.0	338	....
S.M.		92.59	7.41	1.24	6.17	16.7	5.9	136	85
W.W.		91.86	8.14	1.06	7.08	13.0	8.5	155	94
E.E.		96.09	3.91	0.46	3.45	11.7	3.1	131	142
H.H.		91.33	8.67	1.33	7.34	15.27	5.8	282	147
H.H.		90.25	9.75	1.38	8.37	14.10	7.3	255	148
H.H.		89.63	10.47	1.19	9.28	11.38	5.6	256	163
H.H.		89.63	10.37	1.03	9.34	9.97	5.6	246	150
H.H.		90.82	9.18	1.33	8.85	14.50	9.3	.....	....
H.H.		90.16	9.84	1.46	8.38	14.90	9.0	242	147
W.A.		88.44	11.58	1.03	8.92	10.53	9.7	205	90
W.A.		85.80	14.30	1.54	12.76	10.70	7.2	323	96
W.A.		88.90	11.10	1.15	9.95	10.40	6.6	260	126
Average		90.10	9.91	1.12	8.79	10.40	8.9	272	94.4

†Ash is determined as the sulphate.

The samples vary in size from 0.5547 gm. to 3.8393 gm. Ten were below 1 gm. and seven weighed more than 2 gm.

Two reasons for this divergence are seen: *1.* Sinus exudate contains many leukocytes and cellular debris, which we know contains little or no calcium; and *2.* secretions from allergic rhinitis contains a great amount of the protein in the form of mucin. Salvesen and Linder,[6] 1923, from a study of the relation of calcium to protein in sera, chest fluid, ascitic fluid and edema fluid concluded that the calcium decreased proportionately to the protein content. Hastings, Murray and Sendroy,[7] 1926-1927, show a relationship between calcium bound by protein, the protein concentration and pH in human sera. Peters and Eiserson,[8] 1929, have shown a straight line rela-

tionship between the calcium and protein in sera. Arnold and Mendel,[9] 1927, in their study of the interrelationship between the chemical composition of the blood and the lymph of the dog, conclude that interchanges take place between the blood and the lymph, whenever fluctuations in the concentrations of the constituents occur, and that the diffusible substances pass easily and rapidly at all times between the blood, the lymph and the tissues. A study of the values, Table I, for a given individual show that an increase or decrease in calcium is accompanied in most instances by a change in the same direction as the protein. This relationship is disturbed in the pus from sinusitis as the protein content is chiefly made up of cellular debris; however, in mucopurulent discharges from the sinus, this relationship is again indicated, Table III. With clinical improvement of the patient the amount of mucus increased along with the increases in calcium content. We also know from previous work,[4] that the pH in allergic rhinitis is higher than that of purulent maxillary sinusitis. It is well known that serum proteins bind more base at higher pH values. How great a part these factors play in the composition of the secretions analyzed, can only be determined by well controlled experimentation. Also, the permeability of the mucous membrane and the metabolic disturbances must be studied before we can interpret the results obtained in these analyses.

The values of calcium found are much lower than those of Israel,[10] however, the range of variation is only slightly lower. The present findings are not in accord with the statement of Israel, that the majority of instances, the patient with the most severe involvement and the most chronicity showed a high or noteworthy increase in the calcium content of the pus. In fact, the reverse seems to be true, the oldest, most chronic cases have the lowest calcium values with a relatively higher sodium and potassium content. Many more cases must be evaluated before definite conclusions can be drawn.

Blood calcium was determined on patients suffering from allergic rhinitis and sinusitis. The average calcium value was slightly higher in the allergic individuals.

Table IV is a resumé of the cases analyzed. The minimum, maximum, and average percentages of each element determined in the ash are shown. The percentage of calcium is

TABLE III. THE PERCENTAGE OF CALCIUM IN THE TOTAL SOLIDS OF EXUDATES FROM SUPPURATIVE MAXILLARY SINUSITIS.

Patient	Date	Calcium %	Exudate	Treatment
M.H.	2/15/34	0.143	Mucopus	Saline Irrigations
M.H.	2/21/34	0.113	Pus	Saline Irrigations
M.H.	2/23/34	0.130	Mucopus	Saline Irrigations
M.H.	2/27/34	0.151	More mucoid	Saline Irrigations
M.H.	3/5/34	0.360	Opalescent mucus	Saline Irrigations
M.H.	3/12/34	.....	Washings clear	Saline Irrigations
T.T.	4/7/34	0.110	Pus	Saline Irrigations
T.T.	4/20/34	0.168	Heavy mucopus	Saline Irrigations
T.T.	5/8/34	0.227	White mucus	Saline Irrigations
T.T.	5/20/34	.....	Washings clear	Saline Irrigations

Clinical improvement is accompanied by an increase in both the mucus content and calcium content of the exudate.

TABLE IV. THE PERCENTAGE OF CALCIUM, POTASSIUM AND SODIUM IN THE ASH OF NASAL SECRETIONS AND SINUS EXUDATE.

Group*	Calcium			Potassium			Sodium			Ash in Solid
	Min. %	Max. %	Av. %	Min. %	Max. %	Av. %	Min. %	Max. %	Av. %	%
A.	0.48	3.14	1.08	4.72	8.12	6.43	14.0	39.2	25.54	47.00
B.	0.31	2.62	0.93	0.50	26.40	7.01	15.7	34.6	25.76	11.13
C.	0.47	1.60	1.03	2.76	20.18	10.09	15.7	34.1	25.18	26.8
D.	0.26	1.91	0.82	2.05	26.40	8.59	15.7	38.4	23.76	13.5

*(A.) Allergic secretions, 29 determinations; (B.) Material aspirated from suppurative maxillary sinusitis, 29 determinations; (C.) Mucopus washings from cases of sinusitis from which the exudate could not be aspirated, 26 determinations; (D.) Includes group (B.) plus the washings that were secured from the cases after aspiration was impossible, 73 determinations.

The average values were computed from the average values of individual cases. This eliminated undue influence of many determinations on one patient.

the greatest in the secretions containing the most mucus, namely, the mucinous discharge from nasal allergy and cases of sinusitis with mucopus. The potassium values are remarkedly constant in secretions from allergic rhinitis, but in cases of suppurative sinusitis there is a tremendous difference, a minimum of 0.5 per cent to a maximum of 26.4 per cent. The former value is from patient R. F., Table II, having a very tenacious, mucilaginous secretion containing some pus, much mucin and only an occasional eosinophile; the latter from patient S. M., Table II, was a greenish-yellow, purulent fetid pus. This shows very clearly that the type of secretion is reflected in the mineral content. Two other patients, D. K. and W. W., Table II, had a serous amber colored fluid drip from

the needle when the antrum was entered. This fluid held potassium in approximately the same amount as in the blood. In the cases just mentioned, the secretions with a low calcium are associated with a high potassium and *vice versa*. There is a striking similarity in the sodium values, when the minimum, maximum and average values are considered, Table IV. The greatest disturbance in the mineral content appears to be in calcium and potassium, the full significance of which cannot be stated until we have studied the permeability of the mucous membrane as well as the chemistry of the exudate in well controlled animal experimentation.

SUMMARY.

Chemical analysis of the secretion from nasal allergy and the exudate from suppurative maxillary sinusitis has been made. The greatest disturbance in the mineral content is found in the calcium and potassium values in suppurative maxillary sinusitis.

The water and ash content of the nasal discharge from allergic rhinitis is greater than that of suppurative maxillary sinusitis.

The total solids and the protein content of sinus exudate is about three times that of the secretion in allergy.

The ash in the solid is much greater in allergic rhinitis than in the exudate from sinusitis.

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Scott and Euclid Avenues.

CLINICAL OBSERVATIONS ON THE INFLUENCE OF THE NASAL SEPTUM ON THE DEVELOPMENT OF THE NOSE AND THE PALATAL ARCH.*

DR. WILLIAM WESLEY CARTER, New York.

In the growth and development of the human body there are two forces in operation continuously from early childhood until the individual dies. The first is *intrinsic* and is due to the inherent activity of the cells in the process of growth. The second is *extrinsic* and controls or moulds the shape and size of the growing part and incidentally influences its function; its action is mechanical.

The first, or cellular constructive force, is inherent in the tissues themselves and is subject to the state of general bodily health.

The variable quality of the second, the extrinsic or moulding force, is so great that there are no two adult individuals in this world of ours exactly alike from an anatomical point of view. The selection of a typical specimen of the human race would be well-nigh an impossibility.

Identical twins, though they may be alike in every respect at birth, cannot be conceded by the *clinician* to remain so up to maturity, in view of the many hazards, both constitutional and accidental, to which they will be subjected before reaching this age.

The *clinician* deals with the physical results of these developmental forces that have been acting in an abnormal direction on the growing tissues over a considerable period of time. His attention is called to the defect only after the damage is done and has become physically obvious. Fortunately, however, it often happens that the rhinologist, noting a tendency to the development of an abnormality in the nose of a young person, is able to reverse the direction of the forces that are causing the defect. By doing this he may ward off

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an approaching calamity in the way of nasal obstruction and possible deformity of the organ that can be corrected only by a surgical operation. If the patient is seen early enough, one may usually secure a perfectly normal part by directing the developmental forces along normal lines.

There is considerable difference of opinion as to the age limit when these corrective operations may be successfully performed without the aid of surgery. In my own general rhinological work and in the correction of developmental nasal deformities I have been able to secure satisfactory results at any time during the period of active development by proper adjustment, aided in many instances by minor surgical measures.

There are so many circumstances that influence the solution of this problem, such as race, heredity, occupation, environment, rate of growth, constitutional conditions, etc., that it is impossible to settle upon any definite age beyond which a developmental deformity may not be remedied by properly directing the moulding forces. The time element varies with each individual case. Upon one point, however, I am sure that we will all agree, and that is that within reasonable limits the younger the patient is when he comes under our observation and treatment the better the prognosis and the less time that will be consumed in securing the desired results.

My views in regard to the forces that result in the development of the nose of the infant, which at birth is almost on a level with its face, into the prominent organ of the adult and incidentally their influence upon the upper jaw and especially the palatal arch, have been drawn from a study of numerous anatomical specimens and from clinical experience, extending over many years in the practice of general rhinology and in the correction of nasal deformities.

In regard to the influence of the septum, in accomplishing this change in the elevation of the organ, I have not been influenced by anything contained in text-books, for, as far as I know, the subject has not been discussed from my point of view.

I am fully aware of the fact that the views of the clinician may sometimes be at variance with the opinions of those who adhere closely to the academic teachings of the strict conformist; this may be true in this instance; for me, however,

"Seeing is Believing." Clinical experience in every field, we must all admit, transcends all other considerations. Knowledge, gained in this way, comes from direct contact with the patient; therefore, it is practical and its value cannot be gainsaid.

Though the perennial question of what is cause and what is effect may be brought up in this, as in every other discussion, I feel that the anatomical and clinical evidence here brought before you will suffice to show that the septum is a factor of paramount importance, not only in the development of the nose, but that its influence extends also to the superior maxilla and especially the hard palate. Therefore, both the rhinologist and the orthodontist should ever be mindful of its possible effect upon their operative procedures.

The development of the nose and its contiguous and allied structures is, to a large extent, a mechanical proposition and may be compared to the construction of a similar figure from inanimate material to which force has been applied along identical lines.

In order to understand the forces with which we have to deal, the adult nose may be considered a protuberance that has sprung from a more or less flat surface and that the process by which this was effected was, to a certain extent, analogous to that of erecting a tent, the septum corresponding to the tent pole and the soft tissues to the canvas. This is a rough analogy. In clinical experience the constructive forces may be affected by constitutional conditions and especially by such diseases as syphilis, atrophic rhinitis, etc.; also by such intercurrent happenings as traumatism, the development of adenoids, etc. It will serve to remind the operator, however, that normally the septum, from earliest childhood, is under continuous vertical tension and that if, during the years of active growth it is relieved of this tension by uncompensated deflection, by traumatism, by operative measures (such as the submucous operation) or by the incidence of destructive disease, there is certain to be abnormal development, if not actual deformity of the nose and palate, and frequently malocclusion of the teeth.

There are some points in the embryology of the region with which we have to deal that interest us, as clinicians, chiefly

from a physical point of view; only these features will be considered.

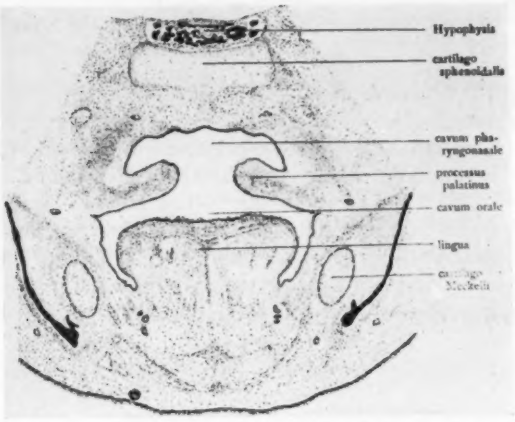

Fig. 1. Primitive vomer; embryo 20 mm. long. Section made near pharynx at back part of the tongue.

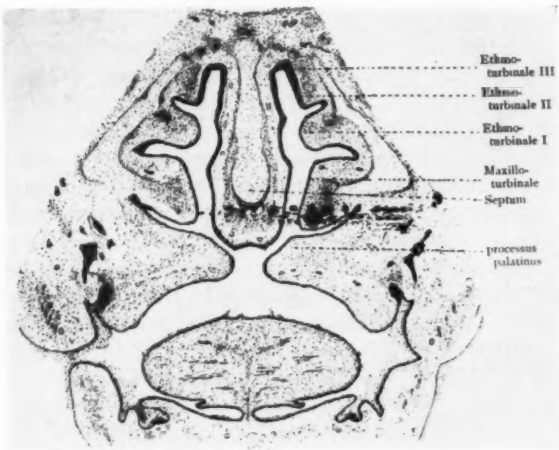

Fig. 2. Section of same embryo, further forward; shows palatal processes forming.

You will recall that the early fetus has only one nasal cavity; this is not separate and distinct, but opens directly into the mouth.

The separation of the oral from the nasal cavity by the palatal processes of the superior maxillae and palate bones proceeds *pari passu* with the division of the latter into the

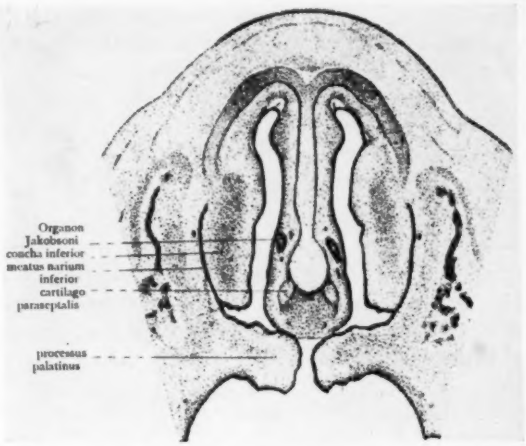

Fig. 3. Embryo 26 mm. long. Shows septum as it is pushing down between the palatal processes of the superior maxillae and palatal bones. It is about to form the tripartite suture.

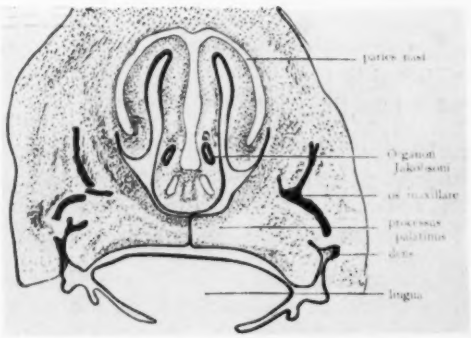

Fig. 4. Section shows the lower edge of the septum in contact with the palatal processes which have united normally in the median line.

two nasal chambers by the primitive vomerine cartilage. A tripartite juncture of these planes in the median line is effected about the eighth week of fetal life. Fusion between the

edges of these three segments, *i.e.*, the vomerine cartilage from above and the two horizontal palatal processes begins forward and proceeds backward; this union is completed about the tenth week.

Ideal development of the nasal chambers, the nasal septum and the palatal arch demands that there shall be perfect synchronism as to the time when the edges of these three planes meet in the median line. This implies that there shall also be uniformity in the rate of fusion and ossification at the line of junction.

It is unnecessary for me to emphasize the delicacy of the problem involved or to express surprise that anatomical symmetry is ever attained. So interesting have I found this situation that for many years I have noted in my patients the appearance of the palatal vault at this suture. I may say, with due conservatism, that in 50 per cent of the patients examined there was anatomical evidence of lack of synchronism, either in the time of fusion or the incidence and rate of ossification. This evidence consisted chiefly in incomplete union (cleft palate), in inequality in the level of the two sides of the hard palate and more frequently, and having greater significance, a bony ridge in the roof of the mouth. This ridge corresponds to the suture and clearly shows that the lower edge of the vomer had reached the proper level for the formation of the suture before the palatal processes had closed in. Consequently, under the vertical pressure, to which the septum was subjected in the raising of the bridge, its lower edge was protruded into the roof of the mouth.

On several occasions I have been called upon to remove such a ridge, for it may cause the patient great annoyance. This operation should be done submucously and care must be taken not to make an opening into the nasal cavity above.

The vomerine cartilage, to which I have already referred, constitutes the primitive septum. It is of interest to note the part it plays in the development of the structure that, in adult life, separates the two nasal cavities.

The vomerine cartilage is a fetal structure, only the anterior portion survives; this becomes the cartilaginous septum. The posterior portion is absorbed after it has accomplished its purpose in the development of the vomer. This is

accomplished in the following manner: About the second month of fetal life a nucleus (osteogenic) appears on either side in the back and lower part of the membrane which covers

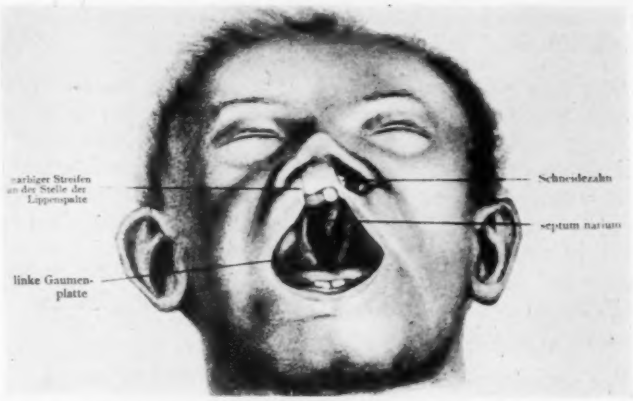

Fig. 5. Cleft palate. Here the palatal processes have not united to form a normal palate. Note the flatness of the nose. This will remain so because the septum has nothing upon which to rest as it tries to elevate the nasal bridge.

Fig. 6. "Wolf nose." A congenital deformity, rarely seen. Here the two plates of the vomer have not united, as they normally should, to form a single bone. There is an interval between these plates reaching up to the base of the skull. In operating upon these cases, extreme care must be taken lest the brain cavity be entered. As usual in these cases, this patient also had a cleft palate. Improvement is the best that can be expected by operation; the defect is congenital and cannot be fully overcome.

vomerine cartilage. From these centers are developed two parallel plates of bone which become fused behind and below about the third month. From this point fusion extends for-

ward and upward, the vomerine cartilage between the two plates being gradually absorbed or pushed forward. Complete union between these two plates of bone is not accomplished until puberty, a circumstance that strengthens my view that the vomer is of great importance in the development of the nose, for the bridge of this organ attains the elevation of adult life about this time.

The vomer may not develop in the normal manner that I have outlined; the bone plates may not become properly fused together, the vitality of the vomer may be impaired or destroyed by traumatism (accidental or operative), constitutional dyscrasias, or by such diseases as syphilis, atrophic rhinitis, etc. If this occurs we may be sure that in every instance imperfect development or deformity of the nose, palate and upper jaw will ensue. The extent of the defect will depend upon the severity of the disturbing factor and the age at which it became operative.

The anterior edge of the vomer lies parallel to the prospective contour of the adult nasal bridge. In erecting this structure, therefore, it has the advantage of transmitting its force at a right angle through the medium of the vertical plate of the ethmoid and the cartilaginous septum. Unyielding resistance to this force is afforded by the hard palate and the base of the skull, so expansion is possible only in the direction of the nose.

Various phenomena connected with the growth of the septum and observable in a large percentage of adults indicate clearly that this structure has been under considerable and long-continued vertical pressure. This evidence consists chiefly in bony and cartilaginous spurs extending along the suture lines between the cartilaginous septum and the vomer, the palatal processes of the superior maxillae and the vertical plate of the ethmoid.

Septal deflections, however, which are seldom seen before the second year, furnish the most convincing evidence of all of this vertical pressure. Deviation of the septum from the median line occurs in 75 per cent of white adults, while it is seldom seen in the Negro and Mongolian races. It is significant to note that the prominent nose is one of the most distinctive characteristics of the Caucasian race; it is evident,

therefore, that here the septum has been called upon to exert its maximum power in the elevation of the nasal bridge, while in the Negro and Mongolian races its effort in this particular has been slight, as is manifested by their broad, flat noses.

The deflected septum shows clearly that in some way it was thrown out of line while it was under pressure and, being unable to recover its vertical position, became more and more deflected as development proceeded.

Many of the conditions that both the rhinologist and the orthodontist have to deal with are due to traumatic injuries received during early childhood and which, at the time, were looked upon as trivial accidents having no important significance and so were soon forgotten.

It has been facetiously said that a child spends half of its life upon its nose. The number of deviated septa, deformities of the nose, palate and upper jaw that one sees would seem to indicate that this is literally true. The deviation slight, and it may be imperceptible at first, becomes more pronounced, consequently the septum loses much of its lifting power and at the same time its force is directed in an abnormal direction. This results in later years in a deformity and nasal obstruction.

Saddle-back noses, twists, lateral displacements of the entire nose and many other unsightly disfigurements are frequently due to injuries that interfered with the normal development of the septum. It should be kept constantly in mind that when the growing septum, which is always under a lifting load, is thrown out of the vertical, it cannot spontaneously recover its upright position. In most of these cases it will be found that the palate is high-arched and that there is protrusion of the upper jaw and malocclusion of the teeth.

One of the distressing features about these cases is that when the patient becomes old enough to realize his disadvantage in personal appearance, he is apt to develop an inferiority complex that is very hard to combat, and which may utterly destroy his happiness and seriously affect his outlook upon life.

There is a close relationship between physical defects and mental disturbance; the situation can be remedied only by removing the cause.

This brings up a difficult question, one that involves much responsibility. What shall we do to relieve a child who is suffering from nasal obstruction due to a deflected septum? Some of the dire results that may follow non-interference have already been enumerated. On the other hand, we know that if the septum is radically removed at an early age a deformity is certain to follow in due course of time. I know that this is true, for I have had many of these cases referred to me for correction.

Several years ago, to meet this emergency, I devised a modification of my original submucous operation. The object of this procedure is to open up the nasal passages and at the same time to preserve the integrity of the growing septum without interfering with its developmental function. Fortunately, I have been able to follow up, over a period of years, some of the results secured by this operation and have found them very satisfactory; there has been no interference with the normal development of either the nose or the upper jaw.

In dealing with this important situation, where the welfare of the child is so intimately involved, the active co-operation of a competent orthodontist is well-nigh indispensable, especially if vaulting of the palate and malocclusion of the teeth are threatened.

In conclusion, I will say that one of my objects in presenting my views upon the subject of nasal development is to impress upon you the fact that there are some very common deformities of the nose, both external and intranasal, that cannot be permanently corrected without close co-operation between the rhinologist and the orthodontist.

There are also dental irregularities, malformations of the upper jaw, especially the palate, that are caused by abnormalities in the nose and throat. Here the orthodontist cannot hope for successful outcome of his efforts until these basic faults have been corrected by the rhinologist.

There is the closest relationship between these two great specialties; their activities overlap and there must be sincere, practical co-operation, actuated by unselfish interest in the patient.

Owing to the efforts that are being made by the Fellows of this splendid Association, this ideal bids fair to be realized,

for its principles are founded upon correct scientific and clinical reasoning; therefore, they are free from personal and professional prejudice.

140 East 54th Street.

MISSISSIPPI VALLEY MEDICAL SOCIETY—

Organized at Quincy, Ill.

A new medical organization to be known as the Mississippi Valley Medical Society was formally organized at Quincy, Ill., on April 8. An annual meeting will be held each fall, devoted to intensive postgraduate instruction and conducted by the leading clinical teachers of the United States. The programs will be eminently practical and of particular interest to the general practitioner.

The Society will especially appeal to the physicians of Illinois, Missouri, and Iowa, and the annual meetings will be held in cities on the Mississippi River in these states. The first meeting will be held in Quincy during the month of October or November and will be a three-day session. The Society has already been approved by the Adams County Medical Society of Illinois (Quincy), and the Marion-Ralls County Medical Society of Missouri (Hannibal).

The officers elected to serve for 1935 are:

President — Dr. Walter Stevenson, Quincy, Ill.

President-Elect — Dr. H. B. Goodrich, Hannibal, Mo.

Secretary-Treasurer — Dr. Harold Swanberg, Quincy, Ill.

Membership in the Society is open to all ethical physicians, it being a prerequisite that all members hold membership in their respective State Medical Societies.

CASE REPORT OF AN INTRACRANIAL ANEURYSM WITH DEATH.*

DR. PURMAN DORMAN, Seattle.

A married white male, age 44 years, tally clerk at the Washington Co-operative Egg Association, was admitted to the King County Hospital, April 26, 1933, with a complaint of pain in his right eye, together with a blurred vision in both, of about seven weeks' duration.

The past history that apparently had much relationship began in 1928, with a bad nose bleed that occurred spontaneously, followed by a succession of nose bleeds that were cleared up by packing, over a course of two years. One severe nose bleed occurred on Jan. 1, 1930. The blood pressure at that time was known to be high and the treatment consisted of packing for the nose bleed, two weeks' bed rest in the hospital and five weeks at home, more or less confined to the bed. Four months after this severe spontaneouss nose bleed, he began to have treatment for his hypertension. He had never had syphilis. In May, 1930, blood pressure was 212 but was brought down to 160 by the use of small tablets, which probably were nitroglycerin. Then followed a period during which he continued treatment for hypertension but there were no symptoms of any sort until March 1, 1933.

At that time he had an acute attack of pain behind his right eye and a blurred vision. After about two weeks the vision in his left eye became blurred but not to so great a degree as in his right. The pain was never so severe as to require treatment until shortly before entrance into the hospital. There was no loss of consciousness at any time and no vomiting. There was a consciousness of pain throughout his illness, spreading so that it seemed to involve the entire left side of his head.

Upon his entrance into the hospital, his first complaint was this pain behind his right eye, blurred vision and a bulging of the eyeball. Physical examination upon admission showed

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*Read before Puget Sound Academy of Ophthalmology and Oto-Laryngology, Seattle, Washington, March 20, 1934.

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a well developed, well nourished, moderately aged man not acutely sick. The pain was localized directly behind the right eye and there were no objective sensory changes to be found in this region. The eyeball was bulging to a moderate degree but the upper lid could be closed over the eye. The pupil was slightly dilated and reacted sluggishly to light. The left eye was essentially negative. Both fundi showed nothing except that in the right the calibre of the arteries was reduced. There was no stiff neck nor Kernig's sign. There was a marked bruit over the right eye, the right frontal, maxillary and temporal areas, synchronous with his pulse, but no thrill could be elicited. The conjunctival vessels were injected while the conjunctiva itself was edematous and insensitive to pain. There was a systolic murmur and his blood pressures, two different readings of which were taken on the same day, were 220/150 and 190/120. After consultation with various members of the staff a diagnosis was made of an aneurysm or an arteriovenous communication, probably between the intracranial portion of the right carotid and the cavernous sinus.

The blood Wassermann was negative. Further study in the hospital added no additional information. Lumbar puncture was not done and his vision was 20/20 in either eye, with the visual fields normal.

Four days after his entrance, under local anesthesia, the right common carotid was ligated. For some 15 minutes before the actual ligation, the vessel itself was compressed by fingers, endeavoring to see whether the lack of blood from this artery would have any immediate effect. Several silk sutures were used to tie it off and the wound was closed. At no time during the operation did the patient experience any cerebral disturbances. At no time after the operation did he become drowsy, suffer any anesthesia or paresis; nor were any cerebral findings noted. Immediately after the operation, the right side of his face was cold but on the following day both sides of his face felt quite warm, and patient felt much better. There was a practical loss of pain, the bulging was reduced and it became easier to close the lids over the bulging eyes. The patient went along in an uneventful manner, with less bulging, less chemosis and less pain until May 5, when it was noted that the left as well as the right eye, which had gone down previously, was not bulging and the conjunctiva

was chemotic and edematous. There was profuse lacrimation in his left eye but no cerebral symptoms were yet noted.

On May 12, thorocatrast, a solution of 25 per cent thorium, was injected into the left internal carotid in an endeavor to delineate the vessel's course and the probable intracranial aneurysmal swellings. Apparently something went wrong because the X-rays that were taken within two minutes showed the solution outside the skull and down in the neck. The internal carotid was sewed up after the withdrawal of the needle. He was again scheduled for an operation but the operating room was running on a close schedule so it was postponed for a few days. Directly after this injection, both eyes were bulging, the left much more than the right, and his vision was 20/20 in each eye, with no changes in either fundus. On May 17, he was again scheduled for operation but, instead of going to the operating room, his condition turned for the worse. He became cyanotic, stuporous and had a cold sweat over his entire body; his pulse being weak and thready. He looked a typical picture of impending death. Something happened, and after eight hours he began to look better and seemed to regain his strength. He was allowed to remain quiet, and on May 23 another notation was made that both eyes seemed to be normal, both fundi were negative, although there was a diplopia of three days' duration. His vision was 20/20 in each eye, with both pupils halfway dilated, with no light reaction, probably due to a sympathetic involvement. There was no bruit. He was discharged from the hospital on May 31, in apparently good health, walking, vision about 20/20 in each eye and apparently entirely cleared from his previous trouble.

We heard no more from him, and a picture taken approximately a month after his discharge showed him to be in apparently good health. About the middle of September, 1933, he began to work at his old job, where he stayed for three weeks. He felt fine during this period until Oct. 19, working until noon that day. He went home for lunch and about an hour after eating he began to have severe abdominal cramps that radiated to his back and to his kidney region, lasting until 6 P.M. About 6:30, he had another sudden acute pain in the midportion of his back, with a gradual loss of feeling in his legs, which began in his toes and spread up to his

hips, followed by an inability to use his legs. After 30 to 40 minutes, he regained feeling in his legs and in a short time was able to walk again. He went to bed early, but he was up eight to ten times, urinating or vomiting. A few times he passed urine that was bloody. Because of poor use of his legs, he fell when he was in the bathroom, striking on his chest and his face.

At this time he was treated by another doctor, in general practice, who was not conversant with his previous history. He was treated symptomatically and his surviving family made but little mention of the previous care that he had had. The doctor who took care of him did not review his past history thoroughly. On Oct. 20th, he was quite drowsy for some 30 to 40 minutes, with a feeling of weakness, a labored respiration and a fairly rapid pulse. He had incontinence of urine. He recovered after about an hour but retained a weakness in his left arm and an inability to raise it, but had no other affected areas. His blood pressure at this time was 226/142, with a urine containing 30 per cent albumin and many red blood cells. His spinal fluid was negative. A diagnosis was made of "hypertension, arteriosclerosis and nephritis." His urinary findings on Oct. 20 showed no albumin and only a few pus cells, while his blood showed 60 per cent hemoglobin. He died on Nov. 5, after a very brief interval of a few hours, when he seemed to be making headway towards recovery. A postmortem was done and the pathologist was not notified of the history during my treatment. As a result, no head autopsy was performed, something that was of the greatest importance to me and for which I had been planning until he got from under my care. The postmortem showed the following findings:

The pericardium was normal. The myocardium showed a marked hyperplasia of the left ventricle, which measures 2½ cm. in thickness. The right heart appeared normal. The endocardium was normal. Five cm. from the aortic valve, there was an opening through the intima and media, from which a dissecting aneurysm extended throughout the course of the aorta, through the diaphragm and along the aorta to the bifurcation, following along the right renal artery, where it had ruptured through the adventitia of the artery with

the blood extravasated into the perirenal tissue and mesentery of the uppor portion of the colon. This caused an infiltration of blood around the vessel, that was 10 cm. in diameter. This was undoubtedly the cause of the sudden death. The right kidney was destroyed, nothing remaining but a retention cyst. This cyst was caused by an obstruction of ureter just beyond the kidney pelvis. This obstruction was a dense scar, the nature of which was not determined. The left kidney was normal. The gastrointestinal tract was normal. The bladder, prostate and seminal vesicles were normal.

Diagnosis: Dissecting aneurysm of the aorta.

Death was due to a sudden extravasation of blood into the peritoneum in the region of the transverse and descending colon.

No autopsy was done on the head.

DISCUSSION.

I believe that we can safely make a diagnosis of an idiopathic developmental type, as compared with an accidental type, of an intracranial aneurysm of both carotid arteries established by the symptoms not only when under our care but also aided by the autopsy, a subjective and objective bruit and the exophthalmos of both eyes. Against the diagnosis of an arteriovenous communication are: *1.* The absence of a whistling type of bruit or of continued thrill or bruit intensified during systole. *2.* The only positive evidence, aside from the operation and actual exposure, would have been examination of blood for the oxygen content obtained from the internal jugular vein at the time of the operation. Blood obtained from the veins near this sort of a lesion has a higher oxygen content as compared with the oxygen content of blood removed from veins situated some distance away from the aneurysm. This distinguishing feature has been suggested by Pemberton,[1] of Rochester. *3.* There was not a great distention of the orbital veins, nor *4.* a marked exophthalmos that would have been more than likely had the cavernous sinus been involved. *5.* The similarity of lesions formed in the autopsy allow us the supposition that a similar thing occurred within the cranium.

It would have been interesting to know what happened inside the cranium at the time he was first operated upon, as well as after his second operation. I believe that at first there was an aneurysm of the intracranial portion of the right internal carotid artery, which was relieved by the ligation. It is pleasing to note that no aftermath appeared of the comparatively sudden tying of the vessel on that side, but following this occlusion, collateral circulation was sufficient to take care of the cerebrum and supply it with sufficient blood to carry on its function. The extra load thrown upon the left internal carotid with its tendency to swell under any load, probably caused a second aneurysm to form on the left carotid, probably at or near the convexity of its last curving within the sinus cavernous at the junction of the ophthalmic artery. This collateral circulation in turn was sufficient to allow the aneurysm of the other side to again swell, so that within about three weeks he had not one but two aneurysms intracranially. As the aneurysms began to swell, pressure upon the eyes became greater, with more bulging and chemosis, until May 17. I firmly believe that on this day, when we thought he was going to die, one or both aneurysms broke, the hemorrhage tending to form a false aneurysm much larger than the original one. That this large aneurysm caused no pressure on the surrounding structures was due only to its position and lack of involvement of structures that presented subjective symptoms.[2] It might have affected the sense of smell by involvement of the olfactory nerve, it might have produced blindness by direct interference with the optic, it might have caused congestion of the eye and swelling by compression of the cavernous sinuses; but nothing occurred, so apparently the second aneurysm caused by the rupture and hemorrhage was not of great amount because we did no spinal puncture but we would have released the intracranial pressure to such a degree that a hemorrhage would have again occurred and death might have come sooner. This man evidently had vessel walls that split easily, and in our particular instance it was much better that death occurred from an aortic aneurysm than from one within the cranium. At the time he was first discharged from the hospital, in 1933, he undoubtedly had the ruptured aneurysm in his head, and death might have occurred at any time during this phase. It was extremely fortunate for him that

he was enabled to go back to work for a short time before he was again taken down. It was a chance circumstance that allowed the aorta to distend and rupture instead of the vessels within the skull. We had a narrow escape when we tied off the right carotid, because, apparently, we had only slight or no damage to the right cerebrum afterward. There was nothing noted after the operation on the left side although, in our closure of the carotid artery after the injection of the solution, the lumen of the carotid was narrowed so that we had a partial occlusion on the left side.

The main interest in the treatment in a case of this sort centers around the danger of a carotid closure. The dangers are mainly two-fold — either a death or permanent cerebrum damage, such as hemiplegia, partial or complete. Writers vary, but apparently there is about 10 per cent mortality; damage usually being due to failure of adequate collateral circulation, most of which comes through the circle of Willis, and arteriosclerosis increases the danger. A factor which tended to minimize the danger of occlusion was the complete rest in bed afterward. A few successful bilateral ligations of this artery are on record. There is one theoretical disadvantage in the use of the common as compared with the internal carotid artery. The external carotid artery has a very free anastomosis with the vessels from the opposite side, which may result in a blood stream coming across and up the internal carotid on the side on which the common carotid has been tied off. As this occurs, the aneurysm then may be refilled through the original route. Neighborhood symptoms, if shown at all, either in the primary or the secdary filling after a ligation, are usually manifest only after partial rupture of the aneurysm and the formation of a false aneurysm.

There are several methods of closing off the vessel.[3] The usual ligatures, either of catgut, silk or linen, provide immediate, complete and irrevocable occlusion with no opportunity to change, should any signs of cerebral damage appear. Halstead and his coworkers, in 1905 and through later years, as well as Matas and Allen in their writings had also become interested in the use of rolled aluminum bands. By this means, partial occlusion was first obtained and if signs of

failing circulation to the cerebrum appeared, the wound was reopened and the band released. If the collateral circulation appeared adequate the band was left *in situ* with a hope of gradual complete occlusion by fibrosis; or the wound might be reopened either to roll the band tighter for complete closure or, if necessary, to remove the band and allow free circulation through the vessel. Matas and Allen advocated a similar technique but used a heavier aluminum band shaped like a letter "U," the degree of occlusion being obtained by compression of the "U." The band could be opened, if necessary, or might be completely compressed, the material being stiff enough to remain fixed. These latter men determined that complete occlusion by this "U" shaped band could continue for 72 hours without damage to the vessel lining and without a thrombosis being formed. In 1911, Neff reported a special aluminum clamp, two bands being hinged to make a "V" with the artery resting between the blades. The free ends of the blades are held apart by plain catgut and the blades are held together by rubber bands. As the catgut absorbs, the blades approximate and compress the artery. Kerr used fasciculator that was not absorbed. Baer's membrane of chromized pig's bladder has been used.

It is unfortunate that a head autopsy was not done for it would have definitely proven or disproved our belief in the previous cranial aneurysms that ruptured. It is unusual to have one series of aneurysms cause one set of symptoms, apparently quiet down as far as general manifestations are concerned, and then have a second aneurysm develop in an entirely new spot and cause symptoms and death without any relationship to the previous history and findings. As we look back upon it now, each was part of the general picture with a tremendous sized ruptured aortic aneurysm causing death.

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THE TRIGEMINOCERVICAL REFLEX.*

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While studying the facial reflexes in patients suffering from encephalitis lethargica and cerebral apoplexy,[1] the attention of the writer was drawn to forward, backward, lateral and rotating craniocervical reflex movements when the nasal or other portions of the face were tapped.

The occurrence of intracranial complications, particularly of the cranial nerves and other structures in the posterior fossa following otitis media, mastoiditis, as well as in luetic, neoplastic, traumatic and other affections of the neural structures concerned with the otolaryngeal and ophthalmic functioning, call for a better understanding of the reflexes that might be of diagnostic aid in the same. The above craniocervical reflex reactions might also shed additional light upon nuchal rigidity, Magnus de Kleijn tonic neck reflex reactions and other signs commonly used in intracranial conditions. The knowledge of the extent of the fifth nerve into part of the cervical region of the spinal cord also suggested interesting possibilities of reflex arc formations resulting in craniospinal reaction phenomena that might also aid in diagnosis of cerebrospinal disease in general.

The tonic neck reflexes of the literature are used mainly as confirmatory signs of pyramidal tract disease. The writer believes, however, that they are ordinary tendon and muscle reflexes, such as the patellar and others, but their presence in the normal has not been emphasized because of their location, and therefore the reactions elicited with them seem to differ from those noted in the other reflexes. Upsetting the equilibrium of the head upon the atlas seems sufficient to do that. The evident relation of not only the pyramidal system but also of the cranial nerves to these reflexes therefore need some emphasis.

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NEUROANATOMY AND NEUROPHYSIOLOGY.

The trigeminal nerve corresponds with spinal nerves in having a motor and sensory root, with a ganglion developed on its sensory root.[2] The widespread distribution of its sensory division is shown by its extent from the sensory nucleus on the homolateral side, where it is in close contact with the Substantia Gelatinosa of Rolando down to the second cervical metamere. Communicating fibres cross all along this path to the opposite side, whence fibres ascend to the optic thalmus, from which another neurone terminates in the post Rolandic cerebral cortex.[3] The relation between the central trigeminal and the pyramidal tracts has also been shown.[4] It is also proven by Rasmussen that the sensory division of the fifth nerve is in close contact with the rubrospinal fasciculus,[5,6] and that the ventral central or secondary trigeminal tract is located posterior to the pyramid and anterior to the fasciculus tectospinalis.[5] The trigeminus is also connected with the cerebellum by fibres constituting part of the direct cerebellar tract and also by fibres passing from the sensory terminal nucleus to the cerebellum as part of the nucleocerebellar tract.[4]

That the descending sensory division of the trigeminal nerve is in intimate contact with the cells of the anterior horn of the first two cervical metameres, thus establishing a reflex arc, is furthermore proven by the following facts: Fibres from the sensory nucleus of the fifth nerve pass down into the spinal cord, giving off collaterals arborising around cells of the substantia gelatinosa of Rolando. The substantia gelatinosa extends from the upper level in the brain to the level of the upper cervical cord, where it makes a cap of cells on the tip of the posterior horn.[3] It consists of two types of Golgi nerve cells, Type I being present in all portions, while Type II is restricted to the dorsal gray column. The Golgi Type II cells send their axones ventrally from the dorsal gray column toward the ventral gray column, many of them entering and ending in this portion of the cord. Many of the axones from these cells cross to the opposite side through the gray commissure.[7] From the above it is evident that certain lesions of the brain and spinal cord must produce changes in the reflex reactions of this anatomically established craniospinal reflex arc. The spinal efferent com-

ponent of this reflex arc furthermore terminates with the cells of the anterior horn, the fibres of which ends in muscles whose function it is to produce flexion, extension, rotation and lateral movement of the head.

Flexion of the head is produced by the rectus capitis anterior and the hyoid group,[8] which are innervated by fibres of cells extending from the first to the fourth cervical metameres.[9] Extension of the head is produced by the splenius capitis, longus capitis, complexus, obliquus inferior and rectus capitis posterior major and minor muscles.[8] Lateral motion of the head is produced by the splenius capitis, longus capitis,

Fig. 1. Method of eliciting the trigeminocervical reflexes.

semispinalis capitis, obliquus capitis superior and rectus capitis lateralis muscles. Rotation of the head is produced by the splenius capitis, complexus, obliquus capitis superior and inferior, and rectus capitis posterior major and minor muscles.[8] The nerve supply of all of the above groups of muscles is located largely within the first two segments of the cervical division of the spinal cord.[9] The anatomical relation of the trigeminal to the first two cervical segments of the spinal cord suggests that the reactions to be described are not due to stimulation of the cervical reflex arc alone, but to their combined action with the trigeminal and other cranial nerves.

TECHNIQUE.

The elicitation of the reflex reaction is illustrated in Fig. 1. While one hand is palpating the muscles of the back of the neck, the other is used in tapping the nasal region, the forehead or other parts of the face. A contraction of the muscles may be felt by the palpating hand. This hand is then shifted laterally, anteriorly or to the opposite side while the tapping is carried out in one locality. In the normal individual palpation is often necessary to confirm the presence of a contraction of the muscles when the reflex is being elicited, particularly when the forward or backward movement of the head is not pronounced. In some pathological conditions, however, the head movements are so pronounced that palpation is not necessary. The palpatory procedure is necessary if one desires to determine whether the reflex reaction is due to stretching of the neck muscles subsequent to a change in the position of the head, caused by the blow of the percussion hammer or to the stimulation of the periosteum. In the former the head is not held fixed, while in the latter event the head is prevented from moving by an assistant while the operator elicits the reflex and detects the reaction by palpation of the neck muscles. The unsupported reaction is probably a combination of a periosteal and stretch reflex.[10,11,12,13] A retraction of the head may also be noted when the masseter reflex reaction is being elicited, or when the cervical muscles are tapped directly.

RESULTS IN THE AVERAGE INDIVIDUAL.

The writer studied the above reflex and found it to be equal bilaterally in over fifty persons who were free from diseases of the nervous system. Persons suffering from osteoarthritis of the cervical vertebrae or myositis of the neck muscles were, for obvious reasons, excluded.

RESULTS IN SICK INDIVIDUALS.

In the following cases examples of segmental cervical reflex involvement of the first two metameres of the spinal cord will be presented. It will also be shown how diseases affecting some of the cranial nerves, particularly the trigeminal and facial nuclei, produce reflex reactions of these craniocervical reflexes that vary from the normal. The abnormal tri-

geminocervical reactions observed in disease of the pyramidal tracts will furthermore be shown in patients suffering from lesions of the same.

Case 1: W. L., an adult, white, male, age 38 years, was seen in the Neurological Wards of the City Hospital. He complained of wasting of his legs and weakness of about five years' duration. He first noticed twitchings in the muscles of his arms in 1918. His past personal and family histories revealed nothing relevant to his condition.

Neurological examination showed no coordinative, sensory, cranial nerve, and no pyramidal tract disease. There were fibrillary twitchings in the upper and lower extremities. The musculature of the cervical region and of all four extremities presented varying degrees of atrophy. Most of the tendon and muscle reflexes of the lower and upper extremities were absent. The blood Wassermann was negative and the spinal fluid examination was also normal.

A diagnosis of progressive muscular atrophy was made.

When either side of the nasal region was tapped with a quick blow of the percussion hammer the resulting movements of the head were not unlike those that may be observed when an empty jug is suddenly upset by an impact that causes it to rotate and finally remain upright. The head swayed rapidly, first to one side, then to the other, rotated, then snapped forward and finally assumed the vertical position. This reaction was noted in two other instances of spinal cord disease, accompanied by atrophy of the cervical musculature such as may be seen in polioencephalomyelitis.

In the following case it will be shown that these cervical reflexes may be affected by meningeal involvement of the base of the brain and associated structures.

Wechsler[14] believes that the tonic neck reflexes of Magnus and de Kleijn are not directly due to meningeal involvement. The anatomical description of the relationship between the sensory division of the trigeminal and the spinal reflex arc of the neck muscle reflexes and the fact that the meninges are supplied by the trifacial nerve[15,16] and others[17] explain the findings in Case 2. The nuchal rigidity of generalized meningitis should therefore hereafter not be taken as a mere pathological clinical sign, but be understood in terms of cranio-

cervical reflex terminology considering the meninges as containing the afferent and the nuchal musculature as being supplied by the efferent components of the reflex arc. The central portion of the reflex arc extends from the sensory nuclei of the fifth down to and including the cellular elements of the substantia gelatinosa to the anterior horn of the upper two cervical segments of the spinal cord.

Case 2: L. G., a married white woman, age 41 years, came to the Neurosyphilis Division of the Neurological Institute in the Vanderbilt Clinic, complaining of a sensation of coldness in and a paralysis of the right side of the face of about five months' duration. Shortly after the above set in, she began to experience a noise in the right ear as though steam was being blown there. This was followed by blindness of the right eye, right frontal headache, swelling of the right side of the face and taste disturbance on the right side.

The history further revealed that her husband died of hasty consumption and that an only child died six weeks after delivery. The cause of death was unknown. Her present illness began six months later.

Neurological examination disclosed a positive Romberg, some weakness in the right hand, normal tendon reflexes, exaggerated muscle reflexes in the right forearm, negative Babinski and confirmatories, and no sensory changes. Of the cranial nerves the first showed some involvement since there was a diminution of the right eye with disc pallor. The right pupil was larger than the left and sluggish to light and the left was irregular, contracted and also sluggish to light. The right masseter was weak and there was a diminution touch and pin-prick sensation over the second division of the trigeminal. The right external rectus was paralyzed. There was also a complete paralysis of the right facial musculature with edema and loss of taste on that side. Hearing was markedly diminished in the right ear, which showed a bone conduction that was greater than that of air. The palate and uvula were moved slightly to the left. The right sternocleidomastoid and trapezius were weak and this condition in the latter simulated an inequality in the scapular reflexes,[18] the muscles of which the trapezius overlies. There was an atrophy of the right side of the tongue.

X-ray and encephalographic examinations were negative. The blood Wassermann was 4+. The spinal fluid contained 29 cells per field with a 94 per cent lymphocytes. The quantitative protein showed a rise above normal and the colloidal gold curve read 1,122,210,000. The urine was negative.

The diagnosis was syphilitic basilar disease of the right side of the brain, particularly involving the right cerebellopontine angle.

In this patient tapping of the right facial region gave no reflex responses,[19] and the right opticofacial[20] as well as the acousticofacial[21] winking reflexes also showed no reactions. Tapping the left side of the face gave a marked contraction of the facial musculature of that side, accompanied by a marked retraction and tilting of the head to the left side. A similar response, *i.e.*, a lateral and posterior movement of the head to the side opposite that of the lesion upon tapping the nasal region of a hemiplegic has been previously reported.[22] In the case of the hemiplegic the reaction was due to the lesion in the pyramidal system, while in this case it was due to an absence of response on the side of the lesion. Many cases such as the above with involvement of the basilar structures alone, due to syphilis or other causes, were seen. The reactions varied in intensity, depending upon the extent of the lesion. These cases differ from the group exemplified by Case 1, in that the involvement did not extend to the anterior horn cells of the cervical metameres, but affected the upper nuclear portions, *i.e.*, the cranial division of this craniocervical compound reflex.

The writer examined about fifty individuals suffering from encephalitis lethargica. From the pathology of this disease it is evident that the nuclear portions of the upper division of the reflex arc of the craniocervical reflex is involved. Unlike the pathology of basilar meningitis where the peripheral parts of the cranial nerves are also affected, this disease is limited to the neural elements situated near the ependyma of the aqueduct of sylvius and fourth ventricles, including largely the nuclei of the midbrain pons and medulla. The reflex reactions in these cases were very interesting since the intensity, rapidity and extent varied with that of the pathological involvement as judged by the disturbances in function and other evidences of cranial nerve disease. Par-

ticularly in cases with oculogyric crises and turning of the head palpation of the nuchal musculature on the part of the examiner disclosed reactions even in muscles considered rigid, as the following case will show:

Case 3: R. P., an adult white female, age 25 years, came to the Neurological Clinic of the City Hospital, complaining of attacks during which her eyes would assume an upward gaze and her head would turn backwards and upwards, with her chin pointing to the right side. The onset of these was one year ago and the duration and frequency progressive so that the spells now lasted for a half hour.

The past personal history disclosed that ten years ago she had influenza followed by a pneumonia and pleurisy which left her in a nervous state so that her face would often twitch, particularly when she became nervous. She was married and had one child. The family history was irrelevant.

Neurological examination disclosed an extremely nervous individual during an oculogyric crisis, with her eyes fixed upward and outward, and the face turned in the same position to the right. The eyeballs presented a constant nsytagmoid movement of the rotary type when the head was in this position and even while she was induced to hold her head in the normal position for a short time. There was some facial inequality on volitional motor functioning, but laughter showed a normal bilateral facial reaction.[22,23,24]

Tapping the facial region showed a greater reaction of the zygomatics and a more marked drawing upward of the lip on the right side when the levator labii superioris propruis was tapped.[25] The above reaction, as well as the downward movement of the lower lip when the lower border of the mandible was tapped,[26] were recently confirmed by other investigators.[27] Otherwise the neurological examination was negative. The diagnosis was lethargic encephalitis.

The craniocervical reflex reactions differed particularly on palpation in the nuchal region where the right side gave a much stronger contraction of the muscles. In this case, as in a number of others, the trigeminocervical reaction was likewise elicited when the patient's masseter reaction was attempted, *i.e.*, there was a retraction of the head in addition to the upward movement of the lower jaw.

In addition to the above, the writer examined over 200 patients suffering from cerebral affections. Many of these cases have been previously reported in connection with work on other reflexes.[28,29] In most of these cases of supranuclear involvement, *i.e.*, pyramidal tract disease above the location of the cranial nerve nuclei involved in the above reflexes, the differences in the cranionuchal reactions between the right and the left side was by far less than that observed with pontine, bulbar, or in the cervical region of the spinal cord.

The above findings suggest the following conclusions:

SUMMARY.

1. Nasal, sinus and pharyngeal diseases as well as conditions of the auditory system often result in intracranial complications.

2. Early diagnosis of such is often important from the surgical standpoint.

3. The meningoencephalites, brain abscesses and other conditions in which encephalography is contraindicated call for clinical signs that would be of more definite localizing value than those heretofore presented in dealing with disease of the posterior fossa.

4. It is therefore important for the otolaryngologist to understand the mechanism of the cranial nerve reflexes that serve as signs of disease in these parts.

5. The trigeminocervical reflexes have heretofore not been reported in the literature.

6. The extensive neuroanatomical connections of their reflex arcs with supra and infranuclear structures seem to offer promising diagnostic possibilities.

7. In supranuclear lesions with little or no involvement of the faciocervical structures there is a slight difference in reaction when both sides of the nose are tapped.

8. With scattered bilateral involvement of the nuclear structures such as noted in lethargic or luetic encephalitis, there is an uneven reaction, depending upon the severity of involvement.

9. In cases of unilateral pontine or pontobulbar lesions there would be found a marked contraction and a throwing

of the head towards the side of the lesion or away from it depending upon its irritative or destructive nature.

10. In patients who have suffered scattered atrophy of the cervical musculature such as observed in progressive muscular atrophy, polioencephalitis, etc., the reactions seem to be the most interesting. There is a rapid rotary backward, forward, sideward repeated rotation of the head with a final whiplike propulsion such as noted in the snapping action of long necked animals.

11. As in other tendon reflex reactions conditions such as myositis, arthritis and local deformities interfere with the proper elicitation of the above signs.

12. The above not only explains nuchal rigidity of meningitis, as in other reflexes on a release basis, but also presents an additional sign of trigemino cervical contraction when nasal regions of patients suffering from meningitis are tapped, though no nuchal rigidity, etc., are present.

13. The above reflexes seem to touch upon a field of cervical reflex possibilities that will shed some light upon a number of clinical signs of pyramidal tract disease in which manipulations of the head are employed.

14. The problems involved in the relationship between the balancing of the head upon the spinal column and the functions of the various groups of muscles which are concerned in flexion, extension, sideward rotating movements and of the erect posture of the head seem to offer a separate field for the diagnosis of local cervical muscular, bone and spinal cord pathology.

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CRICO-PHARYNGEAL FUNCTIONAL STENOSIS.*

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Cricopharyngeal functional stenosis is a spasmodic condition of the upper esophagus, due to a failure of the cricopharyngeus muscle to relax so that the bolus of food may pass downward into the stomach. The etiological factor in many cases is unknown, but it is a fact that foreign bodies, local benign or malignant disease may be the cause.

Symptoms: Difficulty in swallowing solid foods while liquids pass at once into the stomach. Pain in midsternum region when swallowing; especially solid or semisolid foods. These patients as a rule mention the fact that they have not swallowed solid foods for months. One of our cases reported here states no solid food could be swallowed over a period of a year, while another over a period of two years.

Diagnosis: X-ray as a rule reveals no definite pathology, although if a capsule is taken it may, under the fluoroscope show difficulty in passing the stenotic area.

At esophagoscopy there is marked exaggeration of the usual closure that exists at the cricopharyngeus muscle. The lumen of the esophagus above and below is ordinarily normal in these cases.

Treatment: The etiological factor must be searched for and when found treated accordingly. In the functional cases such as are reported here, the passage of a large esophagoscope left in position for three minutes is usually a cure, but it may have to be repeated a number of times. Following endoscopy treatment patient is placed on bismuth powder and belladonna.

Case 1: E. G., admitted to the Clearfield Hospital with a history of difficulty in swallowing and pain in chest. These symptoms began three years ago and have gradually been getting worse. Is able to swallow liquids well but has been

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unable to take solid foods for the past year. The pain in middle of chest is only present when swallowing.

Physical examination revealed a well nourished female, age about 57 years, with moderately infected tonsils, heart slightly enlarged with a systolic murmur, some varicosities of legs, otherwise examination was normal. Her laboratory examinations were within normal limits. X-ray reveals barium passes to stomach rapidly, while a capsule would not pass below the cervical esophagus. Esophagoscopy revealed a marked spasm at the cricopharyngeus muscle and it was several minutes before the esophagoscope could be passed. The mucous membrane above and below were normal, as was the lumen of the esophagus. That evening she was able to swallow solid foods without difficulty and the pain in chest was relieved. This patient returned on three occasions, time limit being nine to 12-month intervals. Each time esophagoscopy was done, following which she would have relief until another spasm occurred. She has not returned the past 18 months, but a recent report from her states she is feeling fine and able to swallow solid foods without difficulty.

Case 2: H. McG., admitted to the Clearfield Hospital with a history of difficulty in swallowing solid foods over a period of two years.

Physical examination revealed a thin female, with a moderate bilateral enlargement of thyroid, infected tonsils, vaginal relaxation, otherwise examination was negative. Laboratory examinations showed some slight anemia. X-ray revealed that barium passes to stomach while a capsule will not pass.

Esophagoscopy: marked exaggeration of closure at cricopharyngeus muscle. Esophagoscope passes through after slight manipulation. The mucous membrane above and below are normal. Following endoscopy she was placed on bismuth powder and belladonna. The following day she swallowed solid foods with ease. She returned several times over a period of 18 months and the large esophagoscope was passed with immediate relief.

Case 3: M. L., age 32 years, female, admitted as an outpatient, complaining of pain in chest on swallowing and unable to swallow solid foods, which symptoms had been present over a period of eight months.

Physical examination revealed a thin female, with a moderately enlarged thyroid, otherwise negative.

Laboratory examination showed a moderated secondary anemia. X-ray revealed barium passes readily to stomach, while capsule does not pass below cervical esophagus.

Esophagoscopy revealed spasm at cricopharyngeus muscle, lumen and mucous membrane above and below were normal. Patient placed on bismuth powder and belladonna. Has reported several times, stating she has had no further symptoms of former condition.

SUMMARY.

1. Condition is more frequent in the female.

2. Etiological factor unknown in most cases.

3. X-ray reveals no definite pathology.

4. Esophagoscopy gives immediate relief.

FRONTAL SINUSITIS; CONCLUSIONS ARRIVED AT FROM OVER 300 INTRANASAL OPERATIONS.*

DR. ROBERT H. GOOD, Chicago.

Since it is more than a quarter of a century that I wrote my first article on the use of a rasp for intranasal frontal sinus operations (*Jour. A. M. A.*, XLX-753, 1907), I feel it my duty to write again, and if possible clarify some of the erroneous conceptions on the controversy between external versus intranasal operations on the frontal sinus. For the description of the intranasal operation, I refer the reader to the article entitled, "Intranasal Treatment of Frontal Sinusitis," *Surgery, Gynecology and Obstetrics*, p. 115, Jan., 1917.

We find statements made in literature which are not substantiated by experience nor facts, such as A. Putschrowsky[1] refers to intranasal methods as "technically they are dangerous to the life of the patient because one may easily, in carrying them out, injure the lower wall of the anterior base of the skull, for that reason they are fairly seldom used nowadays." Dr. Coakley states that intranasal methods with burrs and rasps will lead to more accidents than when the sinuses are opened externally. On the contrary, William Hill[2] in 1914 operated five cases at that time with the Good rasps and said that he was bound to admit that the technique appeared to be both easy and safe. I presented Dr. Hill with a rasp while a guest at his home for lunch in 1913 and he had operated these five cases by 1914.

Herbert Tilley[3] states "that the cases of chronic suppuration of the frontal sinus which call for external operations are few.

"For reasons already given it seems to me that unless there are definite indications to the contrary, the intranasal operation should be given precedence in our attempt to cure or relieve the patient's symptoms.

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*Read before Chicago Otolaryngological Society, April 9, 1934.

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"Serious complications are much less frequent than with the more radical external operations. I have been practicing this operation for three years and so far have had no fatality nor osteomyelitis. For two years I have not performed an external operation but about 30 intranasal operations without fatality."

One needs only to read the writings of Fletcher Ingals,[4] Max Halle,[5] Norval Pierce,[6] R. F. Harrell,[7] H. P. Mosher[8] and many others to verify Tilley's statements.

It is not the purpose of this article to discuss the medical treatment for acute sinusitis, such as cocaine and adrenalin applied to the hictus semilurais, blowing and sucking the nose, compresses and internal medication, as these are fairly well understood.

Conservatism is the keyword in frontal sinus treatments, but if by conservatism we mean leaving an acute frontal sinusitis alone for one or two weeks without giving the patient relief, that is not conservatism but neglect of our duty. It is in the acute cases where our greatest opportunity lies to give instant relief and prevent our patients from becoming chronic sufferers.

O. A. Lathrope[9] states that an acute case should be left alone for 10 days to two weeks. How often have we seen irreparable damage in less time than that and who can say that when a patient has had a spontaneous recovery he will be free from chronic sinusitis setting in?

Paul H. Reed, of Texhoma, Okla., a pupil of mine, probed 12 acute frontal sinuses during his first year of practice and all were promptly relieved without irrigation.

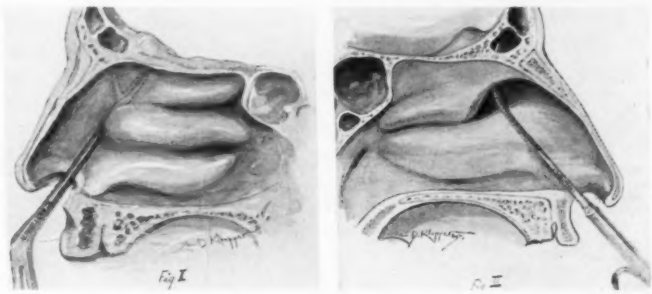
I heartily agree with O. Tilley[10] when he says "an attempt should always be made to pass a cannula into the sinus."

I will go further and state that almost every acute suppurative frontal sinus can be probed, not with a cannula, but with a cotton applicator of proper construction dipped in adrenalin and cocaine. When this has been accomplished the patient will usually find relief whether we irrigate or not. If we now find we can pass a fair sized cotton applicator into the sinus we leave it alone.

In studying anatomical specimens as to the possibility of probing frontal sinuses one must remember that the specimens with very small frontals are difficult to probe and they seldom become diseased. I have never encountered a frontal sinusitis in a small sinus.

O. E. Van Alyea[11] has written an excellent article on irrigation in the treatment of disease of the frontal sinus, which is an excellent treatise on probing the frontal sinus.

Irrigation should only be done when the ostium is large enough to allow free return of pus past the cannula and the cannula should be moved to and fro while syringing. If the ostium is small, suction is much safer. If this procedure does not bring relief in a few days we bite out the ethmoid cells



to the floor of the sinus (see Fig. 1). This latter procedure is indeed seldom required. We insist that an acute frontal sinus must be drained, but with the least trauma possible. I use a cotton applicator which is semiblunt at the end and not too small and fairly strong, so that some force can be used. The cotton must be firmly wound and long enough so that if it should pull off the probe the end would project out of the frontal sinus. A moistened applicator is more efficient than any probe or cannula I know of. The name frontal duct is a misnomer. There is no duct. The so-called duct is simply a passageway between the ethmoid cells.

V. Mueller made a frontal sinus probe (see Fig. 3) for me several months ago, which I have used 12 times and it

may prove to be as valuable as a cotton applicator; this, however, is not enough experience to recommend it.

In the hundreds of cases I have probed I have never been obliged to amputate the middle turbinate. If we encounter a deviated septum which makes the probing impossible, we sterilize the nose and face and perform a submucous resection of the septum to make the probing possible. This would not be done in an office. In chronic cases the same conservative measures are carried out. The opening is made just large enough to establish drainage and relieve the symptoms. One can always make a larger opening later on if necessary. If this conservatism fails we then rasp a large opening at the expense of the frontal sinus (see Fig. 2), but never backward, as the mucous membrane should not be disturbed over more than half of the circumference of the ostium to facilitate the reformation of mucous membrane around the duct. I have simplified my operation from that reported in 1907,

in that I do not remove the anterior end of the middle turbinate except in cases where there is danger of synechia forming between the turbinate and the outer wall.

I have also discarded the protectors as the posterior surfaces of the rasps are smooth and I have discontinued the use of gold or rubber tubes for drainage on account of the discomfort to the patient. I have learned that the least possible irritation and manipulation after operations gives the best results. Occasionally we use our frontal sinus dilators (see Fig. 4) during the process of healing, and when granulations appear we apply a strong solution of silver nitrate to the anterior wall of the ostium with a very small cotton applicator which has been expressed. Of late I am using diathermy in the ostium for granulations and it appears hopeful.

The complications which I have experienced in over 300 intranasal frontal sinus operations without fatality are as follows:

1. Fracture of the lamina paparacia resulting in orbital periostitis (one case).

2. Orbital hemorrhage or hemorrhage in the areolar tissues of the lids (three from injecting ethmoid nerve).

3. Orbital cellulitis (one case).

4. Edema and ecchymosis of lids is rather common.

5. Rasp entering orbit instead of sinus (two cases).

I had one death from meningitis, which was my fifth case, but I used Halle's burr and the burr broke at its attachment to the shaft. This was an accident. This is the first and last time I have used the burr. The motors are so constructed that the burr revolves at a high speed and consequently is very difficult to control. Then again, considerable heat is produced with a burr, which might retard the healing process. It requires more operative space to approach the sinus.

Watson William's[12] guarded burr, *Ibid.* 3-15, is ingenious, but does not overcome the above mentioned objections. Halle has to chisel bone from the outer wall of the nose to make enough room for his straight shaft burr. A mucous flap is not necessary in my operation as I rasp only the ostium.

I will again quote Dr. Herbert Tilley: "I have an intensive preference for an instrument manipulated by the hand and guided by that indefinable but intimate sense of touch." I would like to add to this an instrument guided by the sense of sight, which is accomplished by using a paralleled ray operating light, such as the Kierstein or the late Good light, which is a marked improvement over the former paralleled ray operating lights. All my operations were performed in hospitals as it is almost impossible to be sterile in an office. I have seen a great many operations performed in offices and I have seen the operator use ordinary washed towels, frequently handle the head mirror, turn on lights and touch other unsterilized objects. I have never seen an aseptic operation performed on a nose in an office.

In my early career I engaged a physician for three months, whose duty it was to watch me in my operative clinic to see whether I touched anything whatsoever that was not sterilized, and it was a valuable investment. The temptation or oversight of touching anything not sterilized is far remote from my mind.

INDICATIONS FOR INTRANASAL OPERATION.

From 30 years' experience in frontal sinus work I would say that all frontal sinus cases requiring operation should first be operated by the intranasal route except in the following conditions:

1. Mucoceles, osteomas and other tumors.

2. Meningitis.

3. Brain abscess.

4. Thrombosis.

5. Osteomyelitis.

6. Orbital abscess which has penetrated the periosteum with the pus around the bulb of the eye.

7. Extradural irritation or abscess, the symptoms of which the author refers to in his paper written on this subject in the *Illinois Medical Jour.*, 1919. In these cases the posterior wall of the sinus should be removed, exposing the dura. The outstanding symptoms are occasional subnormal temperature, in the midst of fever, slow pulse, slow respiration, severe pain, which is always in the same place, never shifting. An extradural abscess is the most painful when caused by frontal sinusitis, because of the malpighian bodies in the dura in this area.

8. Orbital abscess with pus between the sinus and the periosteum can simply be drained externally and when the inflammatory symptoms have subsided the intranasal operation will usually allow the fistula to close.

The external operation for acute phlegmon and abscess of the frontal sinus has been followed in my hands with meningitis, osteomyelitis, cavernous sinus thrombosis, general sepsis and thrombosis of the external jugular.

I am not proud of my results with external operations. All my brain abscess, meningitis, osteomyelitis and thrombosis cases have succumbed, but all my extradural irritation and extradural abscess cases recovered. Let me emphasize that frontal sinusitis with symptoms of extradural irritation should be operated immediately and not wait until the infection penetrates the dura, when it is too late to save the patient.

I will refer to a patient that illustrates the effects of both the external and internal operation. Dr. Lynch[13] performed his operation on the left frontal and four months later I operated the right frontal. The technique of a radical operation on the frontal which has given me the best results (100 per cent cures in 15 cases). I saw the movie of this operation at the Chicago Laryngological and Otological Society and in my discussion I censured this operation very severely, for the following reasons:

1. The operation does not permit a thorough examination of the posterior wall of the sinus.

2. If the posterior wall needs removing it cannot be done without great danger of injuring the dura.

3. Diplopia is almost inevitable because the bone to which the pulky of the superior oblique muscle is attached is removed.

4. A suture passed through the orbital periosteum in an infected field entails too much risk of orbital cellulitis.

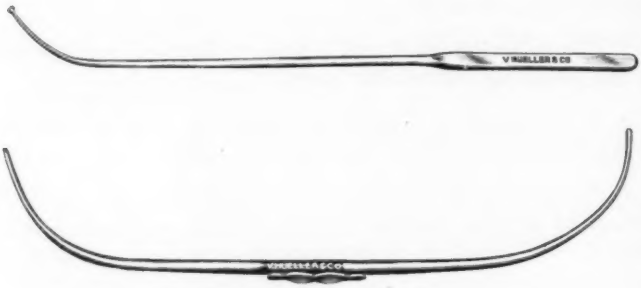
Case: Mr. M. M., age 40 years, was operated on by Dr. Lynch for frontal sinusitis and the patient told me he was in the hospital for four weeks and when he was discharged he was asked to return to have the other sinus operated on. Instead, he came to Chicago to consult the writer. He was wearing a frosted glass over the eye of the operated side on account of diplopia and he stated that he did not want to wear a frosted lens over the other eye with another operation. I operated the other sinus by the intranasal method and he remained in the hospital 20 hours. Mr. M. is a traveling salesman and covers many states and I told him to occasionally see a specialist to irrigate his sinus, and he told me since that he had seen 15 specialists and none of them could irri-

gate the sinus. I could pass the cannula in less time than it takes to write these words.

If specialists do not get better training than this, how can we hope for a general adoption of intranasal methods? Mr. M. is entirely relieved of symptoms from his right sinus, but still has a slight suppurative discharge from his left sinus.

I am convinced that the head mirror is responsible for these failures as I would not for a moment consider using an ordinary head mirror for intranasal work.

Whether we operate the external or internal route, the ostium tends to become smaller by the growth of tissue and bone and it may be necessary in time to reoperate.



The movie which I am showing is on a Miss S. M., whom I operated in 1906. I kept her comfortable all these years, but in the past three years has had spells of retention symptoms and growth of tissue made the ostium very small. If the second operation lasts as long as the first she will not need another, as she will be many years past the three score and ten.

My rasps are right and left (see Fig. 5) with smooth surfaces posteriorly and smooth surfaces on the orbital side. The tips are blunt and the rasp part is long. The rasp is the same width at the base as at the tip.

I conceived the rasp idea from rasps my father made for rasping horses' hoofs and V. Mueller made the first rasp

in 1906. These rasps have not been changed in a quarter of a century as they were constructed for anatomical measurements and never need to be changed. There have bee many modifications made by other physicians and they have changed everything but the handle. They have made sharp pointed rasps and broad at the base. The point could not rasp and the base could not reach the frontal spine. They have overlooked the smooth surface on the orbital side and some the smooth posterior surface. Most of the modified rasps are too short, which defeats the very purpose of the operation, in that there must be enough rasp above the spine in order to rasp.

While I have had no fatalities in over 300 intranasal rasp operations, I do not wish to leave the impression that I have not had much grief. I do not say I have cured all these patients, but I have relieved their symptoms.

I know of no other affliction in our specialty that requires as much good judgment and skill as frontal sinusitis. There is no fixed rule to follow and each patient needs our scrutinizing study.

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IN MEMORIAM

DR. WILLIAM VALENTINE MULLIN

1884-1935

Dr. William Valentine Mullin, of Cieveland, Ohio, died April 25, 1935 of cavernous sinus thrombosis, following a throat infection and an illness of but a few days. He was born in Iowa City, Iowa, Feb. 14, 1884, attended the State University of Iowa and finished his medical studies at Gross Medical College of the University of Denver, in 1908. After doing general practice in southern Colorado he devoted himself to the specialty of otolaryngology, locating in Colorado Springs, in 1912. He did postgraduate work in Berlin in 1914 and came to Cleveland in 1926. He became associated with the Cleveland Clinic as Head of the Department of Otolaryngology, was Secretary and Chairman of the Section on Laryngology, Rhinology and Otology of the American Medical Association, was former Treasurer and, at the time of his death, Secretary of the American Laryngological Association. He was Secretary for postgraduate instruction of the American Academy of Ophthalmology and Oto-Laryngology and a member of the Board of Examiners in Oto-Laryngology. He was a member of the American Otological Society, the American Laryngological, Rhinological and Otological Society, the American College of Surgeons, American Medical Authors' Association, and an editorial collaborator of the Annals of Otology, Laryngology and Rhinology. During the World War Dr. Mullin was a Lieutenant, Medical Corps, U. S. A.

He was a frequent contributor to otolaryngologic literature as is evidenced by more than forty articles. He contributed to the textbooks of Dr. Geo. W. Crile's "Diagnosis and Diseases of the Thyroid Gland" and Drs. Jackson and Coates' "The Ear, Nose and Throat and Their Diseases."

He was a keen and careful scientist, an indefatigable worker, a genial and loyal colleague bubbling over with good humor and there was a real thrill of good fellowship in his handshake. All of these qualities made for his popularity in the various medical organizations with which he was intimately identified.

We mourn his loss greatly and offer our sincere sympathy to his bereaved family.

M. A. G.

THE NEW YORK ACADEMY OF MEDICINE.

SECTION OF OTOLARYNGOLOGY.

Meeting of Nov. 21, 1934.

Dr. Marvin F. Jones, Chairman (presiding). Dr. Wallace Morrison, Secretary

(Continued from March issue.)

Symposium on Upper Respiratory Tract.

I.—The Relationship Between Infections of the Upper Respiratory Tract and the Ear. Dr. James G. Dwyer.

II.—Upper Respiratory Infections from the Pediatric Standpoint. Dr. Louis C. Schroeder.

III.—Endoscopy in the Treatment of Diseases of the Upper Respiratory Tract. Dr. John D. Kernan.

II.—Upper Respiratory Infections From the Pediatric Standpoint. Dr. Louis C. Schroeder.

(To be published in a subsequent issue of THE LARYNGOSCOPE.*)*

DISCUSSION.

DR. JOHN DORSEY CRAIG: I am unprepared and this discussion is entirely extemporaneous. In going over this, I must say that I am not old enough to have any fight with the nose and throat men, because I think after all when we come to the ears that are questionable, where there is some question about opening them, the nose and throat men know more about that than we do. I will qualify that statement and say the nose and throat man who sees the child as a whole, especially the man who works in a babies' ward. He sees the children with the diarrheas caused by infections and in time knows as much as we do about these things.

Speaking of vitamins, I think Dr. Schroeder is right in questioning the dosage. I have been particularly interested in vitamins A and D, especially in reference to the upper respiratory infections as well as for their anti-rachitic value. On the question of maximum dosage for children, we have increased it 800 to 900 times over the dosage advised on the market today with no ill effects. Where the dosage lies that will bring us this desired effect we do not know, but I think the ultimate trend of this dosage of vitamin A in the synthetic carotin and in cod liver oil, and vitamin D in cod liver oil will be that we are now giving from 500 to 1000 times less than we should. A few years ago, of course, there were reports that there might be harmful effects from over-dosage, but we have not reached that stage yet. We do not know where to draw the line, if there is any.

In reference to the tonsils, when to take them out and when not to, I do a lot of teaching and I always like to look at the child's tonsils. Sometimes we are a little lax in impressing upon the mothers and the people who are connected with the child the urgency of removal of the tonsils. Although rheumatism does occur and you do have recurrences and colds do come back, I take the attitude that these horrible things — these chronic cardiacs and chronic nephritis in children — although they do recur, may be caused by infected tonsils. I like to look at every child with an infected pair of tonsils, every infected sinus in childhood and every running ear, with the idea that

this will be one of those hopeless cardiacs and nephritics later on, and so be a little more zealous in taking out these tonsils. I like to work with the nose and throat men. If there is anything questionable in the nose or throat I like to leave it to them. It is the older men who have the fights. We have no fights with them, but turn to them when we have a problem in their field.

DR. SAMUEL KARELITZ: I think that the simplest way to discuss Dr. Schroeder's paper would be to agree with all he has said, because he has told you very important things so well.

The last speaker, Dr. Craig, brought up a point upon which I would like to enlarge. He has mentioned the diarrheas that occur among children who develop an upper respiratory infection. As you all know, the severe cases of diarrhea may become very toxic. In St. Louis they had reached the point where they recommended that children with alimentary intoxication have their mastoids opened because the site of the infection was there. Today these people feel as we do; that is, if the ear is infected in such infants it should be treated as it would be in any other child. Being interested in the gastrointestinal disturbances of infancy and childhood, especially the severe types which go on to alimentary intoxication, I examined the data of 106 cases which I have personally followed in the past five years, to see how many had had upper respiratory infections. I found that of the 106 cases, 41 per cent had demonstrable evidence of infections of the ear, nose or throat on admission to the hospital, while the other 59 per cent showed no signs or symptoms of upper respiratory infection at that particular time. I stress that last statement because it may be significant. I found that of these 43 cases, 28 had evidence of an acute infection of the middle ear, and 15 had what we called dull ears. I feel that this dull ear may be nothing more than a sign of dehydration. Just as the skin has lost its normal appearance with the loss of fluids, so it may be that the drum has lost its lustre, and the dull ear in such cases is not necessarily an indication of something pathological behind the drum. Sixteen of these children had either tonsillitis or an injected pharynx or tonsils without follicular involvement.

It is interesting that several of these children who had no evidences of infection on admission, 11 of them to be exact, went on and developed upper respiratory infections a few days after treatment was started, as their general condition began to improve. I think that bears enlarging upon. Experimentally, it has been shown by Schiff, of Berlin, that if pathogenic cocci are injected intravenously into rabbits, abscesses can be demonstrated in their organs within 24 hours, but the rabbits only begin to show clinical symptoms in four days. If the same thing is done to dehydrated animals they die within 24 hours and autopsy reveals no signs of a reaction tending to abscess formation. By analogy (which, of course may be wrong), we may ask, can this appearance of upper respiratory infections, as such, three to four days after treatment was started, mean that these children were infected while under treatment, or does it mean that during the state of dehydration they were unable to manifest their infection, but did manifest it later when they began to improve from their dehydration and intoxication. (Three of these children became toxic three or four days after a mastoid operation.)

I think that this probably gives you a fair idea of the relationship of these infections to this particular disease, alimentary intoxication. Unquestionably about one-half of them are produced by upper respiratory infections, and of these the ears may be involved in many cases. It is important to remember that the gastrointestinal disturbance may improve promptly if a bulging ear is treated, but this does not mean that the ears should be treated when otologic indications for treatment are absent.

I would like to enlarge upon the statements of Dr. Schroeder in regard to vitamins. I think we speak too vaguely in talking of the good effect of vitamins in preventing upper respiratory infections. I agree with Dr. Schroeder. We respond to the demands of the mothers that they give their children extra

cod liver oil in addition to the daily quart of milk, spinach and what not, but children's colds come as frequently and as severely as ever before. In other words, an excess of Vitamin A does not seem to stop colds.

I think it is a little difficult to conceive of what Dr. Dwyer has inferred, namely, that we might build up an immunity to ear infections and mastoiditis by ingesting more vitamins. I question it, since we do not know of lasting immunity to streptococci, and as Dr. Dwyer himself pointed out, over 90 per cent of his mastoids are caused by streptococcus hemolyticus.

As a pediatrician I would like to call attention to the following: When an infant is in the course of a gastrointestinal upset, and is crying, restless, running high temperature, and has an associated otitis media, I believe that the bulging drum might be incised before the short process becomes invisible. It is not unusual to see a prompt improvement in the entire illness after myringotomy in such cases.

In like manner, the behavior of the child, its sudden loss of appetite, or becoming sulky, restless and resentful of examination may be just as important in deciding whether a mastoid is ready for surgical intervention, as sudden increase in creamy pus, or slight canal wall changes. The child must not be separated from the ear in the decision of surgical intervention in otitic infections.

I have learned conservatism from Dr. Friesner, but I am sure that he would agree with the above exceptions to strict otologic indications for otologic surgery.

III.—Endoscopy in the Treatment of Diseases of the Upper Respiratory Tract. Dr. John D. Kernan.

(To be published in a subsequent issue of THE LARYNGOSCOPE.*)*

DISCUSSION.

DR. DAVID H. JONES: I think Dr. Kernan has covered the complications very thoroughly, and I would mention only that those patients who have developed an acute thyroiditis where the abscess has ruptured through the trachea are exceedingly difficult cases to handle.

I congratulate Dr. Kernan that it was not necessary to do a tracheotomy on his patient with an abscess of the larynx. My own feeling about the matter is that it is far safer to do a tracheotomy early than to do it in an emergency.

I will illustrate with lantern slides the case of a baby, age 13 months, who inhaled a peanut into the lung, and in whom a tracheotomy was done in an outlying city. I wish to stress the fact that in most hospitals the treacheotomy tubes are too short. In addition to the fact that a short tracheotomy tube was used in the baby, it also had an enlarged thymus. Consequently, when the bronchoscope was passed the baby stopped breathing, and it was necessary to use resuscitating measures. The following day radium packs were applied to the baby, 480 mg. hours, which caused a shrinking of the thymus. Bronchoscopy was then done and the peanut removed. With exception of minor complications the baby made an uneventful recovery.

Our pulmonary complications following tonsillectomy under general anesthesia have lessened materially since de-etherizing all our patients by inhalation of CO_2 and O before removal from the operating table.

Clerf, Smith and Manges have written a number of articles calling attention tion to the relationship of sinus disease to lung lesions in children who gave a history of chronic cough, dyspnea, wheezing respiration, undernourishment, and had been diagnosed as asthma or bronchiectasis. Occasionally a foreign

body was found and the lung lesion cleared up on removal, but the others did not show the same improvement.

As so many were diagnosed as asthma an X-ray study of the chest was made, which showed lesions in the lower lobes; others, changes in the root areas resembling glandular involvement; others showed peribronchial changes causing the smallest bronchial tubes to cast shadows, and these changes were, in the majority of cases, in both lungs, the appearance suggesting infection as the cause, then infiltration, fibrosis and inadequate drainage.

Many cases of chronic pulmonary disease in which there was positive evidence of sinus disease were never suspected, and when found were often neglected.

Infection of the accessory sinuses in the opinion of Clerf is the most common cause of bronchiectasis, and it is present in many asthmatics, especially in children. Sixty per cent of the asthmatics have sinus disease shown by X-ray, and 85 per cent of those with sinus disease show demonstrable changes in their lungs. Jackson states that a wheeze heard at the open mouth does not necessarily mean asthma, but may mean a foreign body, bronchial stenosis caused by mediastinal glands or congenital.

Silverman, of Syracuse, states that an examination of 250 cases admitted to City Hospital with scarlet fever showed that nearly all had involvement of sinuses and with one exception all cases with ear complication had involvement of sinuses on the same side as the ear condition.

M. Solis-Cohen, of Philadelphia, stresses the fact that the best results occur in those cases where the secretions were obtained from the lung by bronchoscopy and a vaccine made, and states that the treatment of the sinuses should be done at the same time.

Many patients suffer from chronic cough, tracheobronchitis, and are much improved after the removal of crusts by bronchoscopy, and it is also indicated to remove membrane in tracheobronchial diphtheria.

The entire treatment of upper respiratory infections in children calls for the co-operation of the pediatrician or internist, the rhinologist and the bronchoscopist.

GENERAL DICUSSION.

DR. LEE M. HURD: I agree with Dr. Dwyer. I want to stress a little bit more the fact that tubal lymphoidal tissue, lymphoid tissue in the fossa of Rosenmuller (the pharyngeal recess) is very often overlooked, especially in children who have had a tonsillectomy and adenoidectomy and who have a recurrence of an O.M.C.A. or O.M.P.A. They get nowhere. I found out, after working with diathermy, that it was possible to expose that area with a Yankauer laryngoscope, to go into the mouth of the tube and pick up each lymph follicle and destroy it, being very careful not to hurt the tubal cartilage. You can go into the fossa of Rosenmuller and destroy the adhesive bands and destroy the lymph follicles. In one adult I found the fossa of Rosenmuller three-quarters of an inch deep. The proof of this is that all of these have been relieved of their recurring abscesses of the ear or their recurring catarrhal ears except one.

DR. JOHN M. LORE: In reference to draining abscesses of the pyriform fossa by trephining the thyroid cartilage, I would like to know how many times Dr. Kernan has done this and with what results. I spoke of that once before, last year I think. I always fear in a case of that kind that one may perhaps get a chondritis with considerable difficulty in the management of the larynx subsequent to that. I cannot quite see the rationale of going in deliberately and making a hole through the ala of the cartilage, knowing what we do know of the larynx, when we have to contend with a chondritis time and again. I understand the point that Dr. Kernan makes, that something must be done. I would rather do a tracheotomy first and go in rather leisurely from above.

The interesting thing about these infections of the larynx is the method of spilling. From experimental work on cadavers and animals I have found that as the infection approaches the posterior end of the ala of the thyroid cartilage, pus has a tendency to spill outside the cartilage. If it spilled posteriorly, it would spill back of the carotid sheath. The general rule has been that if the infection is below or at the level of the larynx, irrespective of whether it spills behind or in front of the carotid sheath, the technique of approach should be through an incision along the anterior border of the sternomastoid muscle. If it is higher up, the external approach is along the posterior border of the sternocleidomastoid.

DR. JAMES G. DWYER: I have nothing further to add, Mr. Chairman, except to thank the discussers. In regard to what Dr. Hurd said, that is the very point I wanted to make: that most of us believe that the infection travels up through the Eustachian tube. In many of the cases we fall down, as the infection is in the fossa of Rosenmuller and not in the tube. I am firmly convinced that it was an osteomyelitis process, and I emphasized this lymphoid tissue around the Eustachian tube. Although I have not tried it, I think the method Dr. Hurd has mentioned would be ideal.

DR. LOUIS C. SCHROEDER: I am very glad that Dr. Karelitz has brought out the question of the relationship of diarrhea to the upper respiratory infections. Those who work in children's hospitals see it more frequently there than in private practice. The question is often raised as to which came first, and sometimes it's not easy to answer. The error to be avoided, of course, is to not forget the rhinitis in treating the gastrointestinal symptoms. Dr. Craig's comments about the possibilities of a combative attitude between two groups is something of which I see no possibility. I think that the younger the pediatrician is, the more likely does he feel that he has a miraculous aptitude for doing myringotomies, and as he gets older he will be very happy to pass the responsibility to someone else.

DR. JOHN D. KERNAN: I think that Dr. Jones' remarks very nicely rounded out my paper because he took up matters which I did not touch upon; that is, the connection of acute infections in the nose with infections of the lung in children. In regard to Dr. Lore's remarks, I am delighted that he brought up the point. I want to say that I have used this method on three abscesses such as those I have described. Two were chronic abscesses which had been incised many times from above and although a fistula could be kept open, they did not get well after two or three months of incising. They promptly got well after the operation of trephining the thyroid cartilage, without any chondritis. The third case was an acute infection of the larynx, which formed an abscess inside the pyriform fossa. That also was drained through the cartilage and no further chondritis resulted, although the trephine opening closed before the abscess was fully drained. It reformed and burrowed toward the other side of the larynx. The trephine opening was then carried around the front to the other side and sufficient cartilage removed to fully open the abscess. It promptly got well without chondritis. So I think I can say, basing it, to be sure, on very limited experience, that this operation can be done without an acute infection of the cartilage resulting.

THE AMERICAN BOARD OF OTOLARYNGOLOGY.

During 1935, examinations will be held in New York City, June 8, in connection with the meeting of the American Medical Association, and in Cincinnati in the Fall, during the meeting of the American Academy of Ophthalmology and Otolaryngology. Prospective applicants for certificate should address the Secretary, Dr. W. P. Wherry, 1500 Medical Arts building, Omaha, Neb., for application blanks.

THE AMERICAN LARYNGOLOGICAL, RHINOLOGICAL AND OTOLOGICAL SOCIETY, INC.

The Forty-first Annual Meeting of the American Laryngological, Rhinological and Otological Society, Inc., will be held in Toronto, Canada, on June 3, 4 and 5, 1935, during the week following the meetings of the American Otological Society, the American Laryngological Association and the American Bronchoscopic Society, in Toronto. The meeting of the American Medical Association will be held in Atlantic City, N. J., during the week following.

The scientific program is already being prepared and those Fellows who are planning to present papers should so notify the Secretary promptly. Twelve papers will be selected for presentation.

Copies of the *Transactions* for the past five years are available to those Fellows who have not already received them, without cost. Application for them should be made to the Editor of the *Transactions*, Dr. Lyman G. Richards, 319 Longwood avenue, Boston.

Will you please note the change of address of the Secretary, Dr. Robert L. Loughran, Sharon, Conn.